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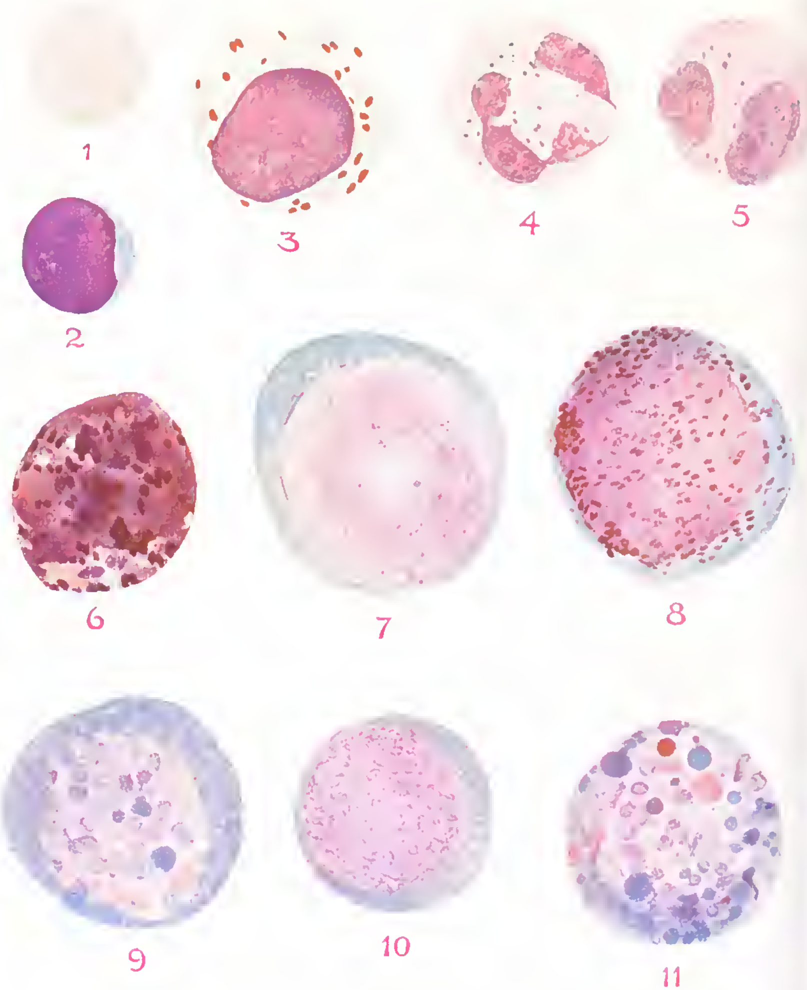
TROPICAL MEDICINE,

HYGIENE, AND PARASITOLOGY.



BLOOD CORPUSCLES STAINED WITH LEISHMAN'S STAIN.

1.—Normal red corpuscle.
2.—Lymphocyte.
3.—Large mononuclear leucocyte.
4.—Polymorphonuclear leucocyte.
5.—Eosinophile leucocyte.
6.—Mast cell.
7.—Abnormal mononuclear, found in trypanosomiasis and certain other diseases.
8-11.—Various myelocytes.

TROPICAL MEDICINE,

HYGIENE, AND PARASITOLOGY.

A Handbook for Practitioners and Students.

BY

GILBERT E. BROOKE,

M.A.CANTAB.; L.R.C.P.EDIN.; D.P.H.; PORT HEALTH OFFICER, SINGAPORE;
FELLOW OF THE ROYAL GEOGRAPHICAL SOCIETY; FELLOW OF THE
ROYAL INSTITUTE OF PUBLIC HEALTH; LECTURER IN HYGIENE,
AND EXAMINER IN CHEMISTRY AND PHYSICS TO THE
MEDICAL SCHOOL OF THE STRAITS SETTLEMENTS;
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Matri Meae Almae,

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MORES ANTIQVOS SEMPER ARTESQVE LIBERALES

NECNON STVDIA NOVA NVPERRIME COMPLEXAE,

HODIERNO DIE OMNIVM QVASI SIGNIFERO EXISTENTI,

QVI, STVDIIS PRAEPOSITI PVBLICIS,

MEDICINAE RATIONEM AVSTRALEM SANITATISQVE

PVBLICAE DOCTRINAM

PRO PARTE VIRILI CONSILIO OPIBVSQVE FOVERVNT,

D. D. D.

"Take up the White Man's burden—
The savage wars of peace—
Fill full the mouth of Famine,
And bid the sickness cease;
And when your goal is nearest
(The end for others sought)
Watch sloth and heathen folly
Bring all your hope to nought.

"Take up the White Man's burden—
No iron rule of kings,
But toil of serf and sweeper—
The tale of common things.
The ports ye shall not enter,
The roads ye shall not tread,
Go, make them with your living
And mark them with your dead.

"Take up the White Man's burden—
Ye dare not stoop to less—
Nor call too loud on Freedom
To cloke your weariness.
By all ye will or whisper,
By all ye leave or do,
The silent, sullen peoples
Shall weigh your God and you."

KIPLING.

[*By kind permission of the Author.*]

FOREWORD.

THE extent of our Empire, and the facilities of modern travel, tend continually to enlarge the sphere of medical observation, both by the introduction of tropical diseases into British ports and by the emigration of medical men to Colonial outposts.

The disadvantages, however, of carrying much luggage from place to place, combined with the ravages of zoological pests, render it essential that a library should be small but complete; while, for the busy student, it cannot but be an advantage that the ever increasing mass of clinical observation and specialised literature should be presented to him in a handy and portable form. I trust, therefore, that the present volume will need no other apology.

Written during the intervals of an unusually busy and strenuous official life, it has been a work of some magnitude (greater than the small compass of the book would lead one to suppose) to collect, sort out, and piece together a vast amount of data from many sources, and to suitably round off the whole by the addition of items from personal experience and observation, and by illustrations specially produced for the work. It has, however, been a pleasant task, and constitutes a link with a life which I have loved, and with peoples and lands which have become to me most dear.

With regard to the contents of the book, it is hoped that the alphabetical arrangement of the section on Tropical Diseases will prove to be not only a novel but a useful feature, and will obviate frequent reference to the Index.

The remarks on Clothing may perhaps be regarded as heterodox, but are, I cannot but think, based on rational premises.

duction of the book. To the following sources I owe a special debt of gratitude and thanks, and crave pardon if any quotations have been made without acknowledgment:—

System of Medicine, ...	Clifford Allbutt.
Diseases of Warm Climates, . . .	Scheube.
,, ,, . . .	Davidson.
Tropical Diseases, ...	Manson.
Lectures on Tropical Diseases, . .	Manson.
Laboratory Studies in Tropical Medicine,	Daniels.
Practical Study of Malaria, . . .	Stephens & Christophers.
Malarial Fevers, ...	Thayer.
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,, ,, ...	Thomson.
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Plague, ...	Cantlie.
Beri-beri (F. M. S. Research Inst.), .	Hamilton Wright.
,, ,, ,, .	Daniels
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Leprosy, ...	Hansen & Looft.
Yellow Fever, ...	Martin.
Manual of Bacteriology, . . .	Hewlett.
,, ,, . . .	Muir & Ritchie.
Essentials of Practical Bacteriology, .	Curtis.
Mycology of the Mouth, . . .	Goadby.
Diseases of the Skin, ...	Crocker.
,, ,, ...	Stelwagon.
Pathology of the Skin, ...	Macleod.
Public Health, ...	Lewis & Balfour.
Hygiene, ...	Notter & Firth.
Practical Guide to Disinfection, . .	Rosenau.
Diseases of the Blood, ...	Coles.
Insects (Cambridge Natural History), .	Sharp.
Animal Parasites of Man, . . .	Braun.
Gnats or Mosquitoes, ...	Giles.
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The Medical Annual.
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Journal of Tropical Medicine.
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Journal of B.M.A. (*Malaya Branch*).
Publications of the Bureau of Government Laboratories, Manila.
&c. &c. &c.

GILBERT E. BROOKE.

BATH, *March*, 1908.

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TROPICAL MEDICINE.

CHAPTER I.

INTRODUCTORY.

The first thing that will strike the new-comer to tropical regions is the great unlikeness of almost every feature of life to those to which he has been accustomed in a temperate zone.

The tropical type of buildings; the many vivid pictures, like a wonderful kaleidoscope, which will flit before him under the glaring sunshine in a cosmopolitan tropical city; the infinite types of natives, black, brown or yellow, each with their different language, strange garments and unknown mysterious potentialities: the rich and unrecognised flora; the teeming forms of lowly animal and vegetable life; the prevalence of such pests as mosquitoes, centipedes, scorpions; the invasion of one's larder and dining-table by innumerable species of ants, and of one's floors and woodwork by that *bête noir* the white ant; the weird croaking of the bull-frog after rain; the ceaseless chirp of countless cicadæ through the short, hot tropic night; the dancing light of fireflies in the gloom of the dense vegetation; the cheerful antics of the small house-lizard on his normal hunting ground—one's ceiling or wall; the strange fruits and unknown flowers; the weedy, anæmic look of European children—these, and a hundred other features, will soon make the new-comer feel that he has indeed reached a *terra incognita*.

But, though familiarity will soon breed some contempt and much forgetfulness, yet an indefinite charm will usually grow with the passing years; and not only will a love spring up for the sunshine, the unconventionality, the untrammelled freedom of life beneath the tropical sun, but, to the keen scientist and student, there will be an ever-increasing fascination for the many solved and unsolved riddles of tropical medicine.

To the tropical practitioner, mayhap, opportunities (both of time and place and perhaps of natural bent) for original pathological research may often be wanting. I would not go so far as to say, with Manson, that the student of medicine must be a naturalist before he can hope to be a capable practitioner, for any man with such an elementary command of techique and method as is acquired at a school of tropical medicine at home may be admirably equipped

for clinical diagnostics; yet, for the profounder investigations of scientific epidemiology or obscure etiologies, greater laboratory facilities, and more time and money are required than are usually at the disposal of the busy medical official or tropical practitioner.

What can be done, however, with limited opportunities and appliances can be admirably seen in such a book as *Daniel's Laboratory Studies in Tropical Medicine*, which should be in the hands of every keen student.

The more earnest workers and collectors there are in all parts of the world, the more will our knowledge of disease conditions spread, to the consequent benefit of humanity at large. Tropical medicine has made enormous advances in recent years, so much so that a text-book of ten or more years ago seems but a list of grotesque gropings and exploded hypotheses.

Let those who stay at home and think that the subject of tropical medicine is a small thing and unworthy of study as a specialised branch, but come and practice for a year in the tropics, and find themselves face to face, day after day, with symptoms strange to them and pathological conditions beyond their ken—they will then perhaps realise that the host of diseases peculiar to, or especially rife in, the tropics are, to the old familiar home lessons, what the wealth of tropical flora is to the botanist who only knows the delightful hedgerows of the old country.

As our ideas have been revolutionised, and we have gradually come to realise the vital part which is played by insects and other lowly forms of life as intermediate hosts in the dissemination of disease, so now we are met by the question as to how we can best utilise this knowledge in everyday life, and apply it to the investigation of the unknown problems which continually confront us.

Manson's excellent suggestions, given in his *Lane Lectures* at the Cooper Medical College, San Francisco, should be mentioned in this connection. He proposes:—

1. That medical men who intend to practise in tropical countries should be thoroughly grounded in theoretical and applied tropical pathology, and maintains that to send a young graduate to treat tropical diseases he has never before seen or studied is unfair both to himself and his patients, and is acting with regard to human life in a way that no merchant would think of acting in his business matters.

2. That the *rôle* of insects in the spread of tropical disease should be suitably taught to all Government employees. The necessity for such a measure is obvious. Government employees are expensive; what is spent on administration cannot be spent on roads, railways, and other remunerative public works; anyone, therefore, who by his own carelessness allows himself to be infected is a serious and *avoidable* monetary loss to the Government of the Colony.

3. That the rudiments of tropical hygiene be suitably taught to all Government or other native schools. More good can thus be done in the cause of sanitary prosperity than by innumerable hard-

working medical officers of health and their inspectors, for it is the native school children who in a few years will be the adult population of a place; and they will accept, as a matter of course, sanitary

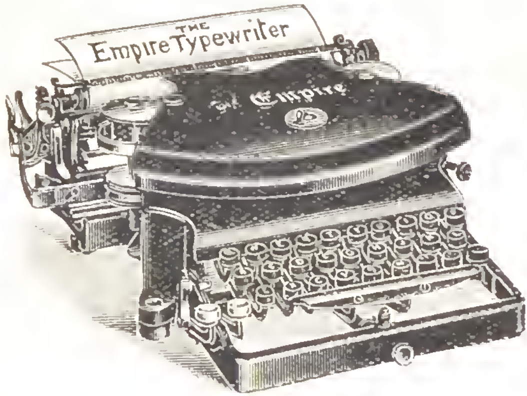


Fig. 1

measures which their parents would have rejected with suspicion or contempt.

Many problems of tropical medicine are urgently awaiting solution, and it behoves every tropical practitioner to do his utmost towards their elucidation.

A first-class microscope is an essential of any research work, and a good camera is almost as necessary. Hints on these instruments will be found in Chapters xxxix. and xl.

If much writing is contemplated, such as notes, contributions to journals, &c., of which copies have to be kept, then a good typewriter will prove a great boon. Almost any typewriter will answer the purpose, and all have many points of excellence; but perhaps the one which most combines simplicity of construction with compactness and portability is the "Empire" (Fig. 1), and these are points which the traveller cannot afford to overlook.

A few concluding remarks on the care of apparatus, books, &c., will not be out of place:—

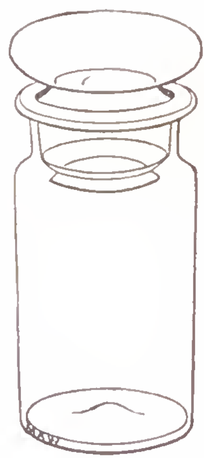


Fig. 2.—Bottle.

In the tropics there is a special form of mould which attacks glass, making a branching fern-like pattern which defies either hot or cold alcohol, acids, or alkalies when once it has appeared.

To obviate this calamity *lenses* of all kinds should be wiped with a soft cambric rag at least twice a week, and kept in as dry a place

as possible. An alternative is to keep them in a drying jar or box. Such receptacle should be either a wide-mouthed glass jar with a store of quicklime in the lid (Fig. 2), or else some form of box, cupboard, or glass case, under the perforated false bottom of which is kept a dish of calcium chloride. This dish can be put on the kitchen fire whenever it is necessary to re-dry the calcium chloride.

Photographic plates and paper should all be kept in a drying bottle or box, or they will quickly deteriorate. *Microscope slides* should be kept in alcohol; and *cover slips* either in alcohol or carbolised glycerine.

With regard to *books* it will be found that in places with a high relative humidity it is very hard to keep them free from *Mucor mucedo* and other moulds; moreover, the linen or leather bindings are eaten by cockroaches, weevils, white ants, rats, &c.

To obviate this the author has found it a good plan to paint all books once a year, or more often if necessary, with the following varnish :—

The Book Varnish.—Dissolve ½ oz. of shellac in 8 ozs. of methylated spirit, and add 2 drams of creosote. Shake well before use, and apply evenly with a flat brush.

This will be found most efficacious in keeping zoological pests at bay, and has the advantage of drying in a few minutes.

Another point to notice in exceptionally humid climates is—never keep books in a closed bookcase unless they are wiped weekly, and the bookcase aired in the sun. Otherwise, whatever precautions are taken, the books are apt to get mouldy.

Mosquito-bite Prophylaxis.—The new comer to the tropics, with fresh, vigorous circulation, is a god-send to the blood-thirsty mosquito, who makes the best of his opportunities to the great grief of, and often danger to, the person so operated on.

Exposed situations, such as the ankles, neck, hands, &c., are, of course, the favourite spots; and especially the ankles, unnoticed under a dining-table.

Many applications have been suggested, some of which are pleasant to the smell, and others disagreeable; and, generally, their virtue only lasts for a limited time. The following is a short list:—

1. *Citronella Oil.*—Apply to skin in very small quantities. Pleasant smell. Effect lasts for an hour or two.
2. *Eucalyptus Oil.*—Apply as above.
3. *Tincture of Pyrethrum.*—Dabbed on to exposed parts.
4. *The Prophylactic.*—

Ol. Pini Sylvestris	
Ol. Amygdalæ, āā, ...	ʒvj.
Ol. Mentha Pulegii, ...	ʒiv.
Spir. Camphor, ...	ʒiij.
Glycerine, ...	ʒijss.
Ac. Carbol. liq., ...	ʒi.

M. Shake before application.

5. *Antikito Cream.*—Obtainable from Antikito Syndicate, Ltd., Great Portland Street, London.

Notes on Tropical Practice.—The practice of medicine in warm climates is a subject which, by a Committee of the Royal College of Physicians, was only recently pronounced as being too small to merit the distinction of a special diploma.

To the tropical practitioner the matter will appear far otherwise. Not only is he surrounded from the outset by a veritable host of pathological conditions which are new to him, but he will find that they are often dependent on flora or fauna previously unknown, that the physiological functions are all exaggerated, that the problems of sanitation are entirely novel—in fact, that tropical medicine is the study of a lifetime, the study of a thousand problems of which the interest and far-reaching importance are incomparably greater than are those of the well-beaten track of medicine in temperate climates.

Perhaps one of the first features that will strike a practitioner in the tropics is the frequent occurrence of fever. At home he has always been taught that fever is a symptom due either to some grave specific toxæmia or to some inflammatory process. He comes across case after case of pyrexia; there is no hint of anything inflammatory. He thinks at once of malaria, and examines the blood and gives quinine, without avail; he excludes the zymotic diseases, and then sees that his ideas need revision, and that he has to deal with a tropical febriculum—a manifestation of a strained physiological mechanism.

Again, he visits a patient, obviously seriously ill with high fever, laboured breathing, and a bad pulse. He finds some moderate areas of lung dulness. The patient dies after only two days' illness, and he signs it up as pneumonia. A few days later he is surprised to find five or six people in the neighbourhood seriously ill with high fever, a full, fast, and low-tension pulse, an anxious expression, and foul tongue. He may think of typhoid, and, in exposing the abdomen, discovers some tenderness in the groin leading to the disclosure of a large bubo, and an explanation of the speedy death, in the pneumonia, which he had overlooked.

Perhaps, again, he is a ship's surgeon on a vessel carrying Coolies. The day before entering port a sudden death occurs, and the same evening a case of bad diarrhœa, which dies, after perhaps one or two doses of opium, or of chalk and bismuth. On arrival in port he reports the two deaths—one as heart failure and the other probably diarrhœa in an "opium smoker"—since the patient had appeared very emaciated. He is perhaps highly disgusted to find that the Coolies are quarantined—an action which is justified by a subsequent smart cholera epidemic.

These are but a few instances of mistaken diagnoses which would generally be obviated by a study of tropical medicine combined with practical experience.

Other awkward points may erop up besides these diseases peeuliar to the tropies. The praetitioner may be ealled on to examine for *life assurance;* he should know in what way the probability of life is altered by previous tropieal diseases.

He may be summoned to treat a ease of *snake bite* or of *poisoning by native drugs, &c.;* it will therefore be neeessary for him to have some knowledge of these points.

Then, again, with regard to *furlough*, it is often a diffieult matter to deeide (even for the experieneed praetitioner) whether a patient ean with safety be treated in the plaee itself, or whether he should be sent home on leave.

There are other eases, too, in whieh there is no aetive illness, but in whieh furlough is indieated. Negleet of these adverse signals may lead to a rapid breakdown and perhaps a lengthy ineapaeity for work, which eould easily have been obviated by a little timely eireumspeetion.

The following earefully eompiled table may in some sort serve as a guide in the multifarious eventualities of life in the tropies:—

(*a*) *Indications for short furlough to hill-station, or for a sea-voyage:—*

1. Reeovery from a wild attaek of any infeetious disease.
2. Persistent insomnia.
3. Reeovery from malaria.
4. Reeovery from dysentery.
5. A "toueh of the sun," with irritability, headaehes, and amnesia.
6. A persistent morning diarrhœa.
7. After a hepatitis.
8. Three years in the tropies without leave.

(*b*) *Indications for long leave:—*

1. After reeovery from plague, eholera, liver abseess, or typhoid.
2. After reeovery from severe attaek of malaria, smallpox, dysentery, yellow fever, Malta fever.
3. Spirillar fever.
4. Kala-azar.
5. Diathermasia or phœbism (if severe).
6. Sprue.
7. Bilharziosis.
8. Blaekwater fever.
9. Nervous breakdown, evideneed by several of the following symptoms:—Insomnia, irritability, anæmia, amnesia, anorexia, headaehes, lassitude, exeessive diaphoresis, phosphaturia, reeurrent diarrhœa, &e.
10. Five years eontinuous residence in the tropies.

(*c*) *Continental treatment for tropical cases:—*

1. Tropieal anæmia with nervous breakdown should be treated by a rest eure at *Schwalbach.*

2. Goutiness, renal inadequacy, abdominal plethora, and similar cases do well at *Evian*.
3. Convalescence from malaria or malarial cachexia should go to the iron waters of *Spa*.
4. Hepatic indequacy, tropical liver cases, and other gastric and hepatic cases should undergo a course of treatment at *Brides-les-Bains*, followed by *Prolagnon*.
5. Cases of early leprosy should try the rich chloride waters of *Carlsbad*.

The settler, the traveller, the merchant whose occupation or pleasure necessitates a tropical life, often find themselves out of reach of medical aid. This is replaced in many cases by a private medicine chest and some manual, such as *Hints to Travellers*, of the Royal Geographical Society; or other similar volume.

Perhaps, largely owing to this circumstance, the use of *patent medicines* is, if possible, even more prevalent in the tropics than at home.

The medical man may be called to a patient, and find him with a serious illness for which he had been taking boxesful of So-and-So's pills. The composition of these quack specifics is often unknown to the medical attendant, who may, therefore, find the following list to be of use, compiled from a Lecture on Patent Medicines by *Hutchison*:—

Antikamnia,	Sod. Bicarb., Antifebrin, and Caffein.
Beecham's Pills,	Aloes, Ginger, and Soap.
Bile Beans,	Cascara, Rhubarb, Liquorice, Oil of Peppermint, coated with Gelatin.
Bromidia,	Pot. Brom., Chloral, Hyoscyamus, Cannabis Indica, Oil of Aniseed, Syrup and Water.
Bunter's Nervine,	Creosote, Chloroform, Camphor, Balsam of Tolu, and Alcohol.
Carter's Little Liver Pills,	Podophyllin (gr. $\frac{1}{8}$) and Aloes Soc. (gr. $\frac{1}{3}$).
Clarke's Blood Mixture,	Active constituent is Pot. Iod. (6 grs. to the oz.).
Cockle's Pills,	Aloes, Colocynth, and Rhubarb.
Doan's Backache Pills,	1. [Dinner Pills] Podophyllin, Aloin, Rhubarb, and Peppermint. 2. [Backache Pills] Oil of Juniper and a resinous constituent, ? Copaiba.
Eade's Pills,	Sod. Salicyl, Guaiacum, and Aloes.
Eno's Fruit Salt,	Sod. Bicarb., Acid Tart., and Acid Cit.
Guy's Tonic,	Acid Phosph., Tinct. Cochineal, Inf. Gentian, and Chloroform Water.
Holloway's Pills,	Aloes, Rhubarb, Saffron, Glauber's Salt, and Pepper.

Keating's Cough Lozenges, .	Ipecac., Lactucaria, Squill, Liquorice, Tragacanth, and Sugar.
Lamplough's Pyretic Saline, .	Acid Cit., Sod. Bicarb., and Pot. Bicarb.
Owbridge's Lung Tonic, . .	Balsam of Tolu, Oil of Aniseed, and Oil of Cloves.
Phospherine, ...	Quinine, Phosphates, and Hypophosphites.
Pink Pills, ...	Sulphate of Iron, an Alkaline Carbonate, and Liquorice, thickly coated with Sugar and coloured with Carmine.
Seigel's Syrup, . . .	Aloes, Capsicum, Liquorice, and Treacle.
Steedman's Teething Powders,	Calomel and Starch.
Warner's Safe Cure, . .	Pot. Nitrate (10 grs. to the oz.), with various Diuretic Herbs.
Whelpton's Pills, . . .	Rhubarb, Aloes, Ginger, Pulv. Ipecac., and Soap.
Woodward's Gripe Water, .	Liq. Mag. Carb., Ol. Anethi, Sugar, and a trace of Alcohol.

CHAPTER II.

CLIMATOLOGY.

THE word climate, from the Greek χλιμα from χλινω to incline, was originally used to express the apparent inclination of the heavens towards the horizon.

By climate we now mean the complex series of meteorological conditions which obtain in any given locality. We have to take into consideration the mean and extreme temperatures, the barometric pressure, the rainfall, and humidity.

The latitude of a place is the chief determining cause of its temperature, although a glance at the isothermal lines in the accompanying chart will show that this statement is only true in a general way; for the real temperatures, owing to other influences, do not exactly accord with latitudes.

The heat not only depends on the greater or less obliquity of the sun's rays, but also on a greater or less column of atmosphere. The column of air is greatest at the sea level. The higher the point we reach from this sea level, the greater the rarity of the air becomes, and the less the heat of the atmosphere due to solar rays.

Other factors also serve to modify the temperature, such as proximity to warm or cold sea currents, or to mountain ranges, the nature of the soil, the direction of prevailing winds, the relative humidity, the amount of vegetation, &c.

We may classify climates in two ways (see Figs 3 and 4).

I.—Climates Classified by Temperature.

1. Warm Climates.—Including tropical and parts of sub-tropical places. These lie chiefly between the Equator and latitude 35° north or south of it. They are characterised by a mean annual temperature of 70° to 80° F., heavy rainfall, marked dry and wet seasons, and luxuriant vegetation.

Equatorial countries, such as the north of South America, Central Africa, and the Malay Archipelago and Peninsula, tend to have the most excessive rainfall, the seasons not being so marked as at a greater distance from the Equator.

Speaking generally, from 5° to 10° there are two rainy and two dry seasons in the year, from 10° to 25° there will be found one rainy and one dry season only. Outside these limits the rainfall will be less, and the rainy seasons again become less well marked.

Certain diseases are usually attributed to warm climates—such as heat effects, plague, cholera. smallpox, yellow fever, malaria, dysentery, dengue, &c. This division is, of course, only arbitrary, but their prevalence in these climates will warrant the classification, which, if carried to its logical basis, will be found to be dependent, not so much on the temperature *per se*, as on the results of that temperature—such as the determination of the vital activity of certain intermediate hosts, and the extremely prolific development of the lower forms of vegetable life, such as bacteria, moulds, &c.

2. Temperate Climates.—Such climates are usually found between the latitudes of 35° and 50°, and are characterised by 4 well-defined seasons, a smaller amount of rain (not limited to definite seasons), and a mean temperature of 50° F.

The diseases of temperate climates are those of every-day English life, and do not call for mention here.

3. Cold Climates.—These are situated between 50° of latitude and the poles.

They have a mean annual temperature of 20° F. The winter is long and the summer short. There is abundance of snow but scarcely any rain.

No special diseases can be attributed to these cold climates, unless we include such as chilblains, frostbite, gangrene, and perhaps scurvy, which is more probably not affected by cold, but only by defective alimentation. In these regions the pulse of life beats slow.

Micro-organisms flourish with difficulty; only the fittest and hardiest of vegetable life can survive. Smallpox, however, is said to have occasionally prevailed in Greenland, and sub-acute and chronic rheumatism and catarrhal chest conditions may occasionally be found.

II.—Climates Classified by Barometric or Hygrometric Conditions.

1. Mountain Climates.—By this is meant an elevation of 2,000 feet or more, and the rare atmosphere which there occurs is characterised by lowered temperature, slightness of relative humidity, much sunlight, and considerable freedom from suspended matter and vegetable organisms. The barometric pressure is low, the barometer falling $\frac{1}{10}$ inch for each 100 feet of elevation above sea level.

The rainfall largely depends on the nature of the land which intervenes between the mountain and the sea, and also on the direction of the prevailing winds. When damp sea winds meet mountain ranges and are forced upwards—*e.g.*, the Western Ghats on the Malabar Coast with over 260 inches yearly, and the Khasia Hills, north of the Bay of Bengal, with over 400 inches yearly, and a reported 805 inches in 1861.

John Bartholomew & Co., Edinr.

Mountain climates are well suited to those with lung affections, hereditary or acquired, such as imperfect chest development or phthisis, but are unsuited for acute stages or chronic bronchitis, or those suffering from kidney, liver, or brain affections—and are also distinctly hurtful to the aged or feeble.

2. Marine Climates.—These should include both *insular and littoral climates* which are characterised by equable temperatures, considerable relative humidity, and bland atmosphere, due to their proximity to the sea.

The principal diseases in any way peculiar to these climates are lung affections and rheumatism, aggravated by the constant weather changes and humidity.

3. Continental Climates.—These exist over the large expanses of land at a distance from the sea. Here the influence of the nature of the soil and amount of vegetation come largely into play. Sandy soils are warmer than clay or compact soils. The equalising effect of the sea being absent, there is a liability to great extremes of heat and cold.

The rainfall is largely regulated by the amount of vegetation. Thus some desert Continental tracts which are devoid of vegetation are practically without a rainfall; such are the Sahara, Arabia, the deserts of Central Asia, South Africa, Australia, and the great salt lake region of North America. Phthisis is very rare in these dry regions.

The ordinary Continental climate has no special disease selection. The great range of Continental temperatures is shown by the following shade temperatures, quoted by Davies:—Werchojansk, in Siberia, has a mean temperature of $-56°$ F. for January. The minimum temperature recorded at this place is $-81°$ F.

In North Africa, at Murzuk, 130° F. has been recorded in the shade; while, in India, Blanford has recorded 123·1° F. at Pachpadra.

The mean solar radiation varies from 130° to 160° F., and has been known to reach 175° F.

The Influence of Climate on Health.

The multitudinous effects have long been recognised, and have become a most difficult problem in regard to schemes of colonisation, movement of troops, or location of colonising centres. Probably many sections of the human race owe their national characteristics mainly to climatic influences continued through a series of generations. The differences between the different members of the Aryan race, such as Celts, Germans, Anglo-Saxons, and Italians, as also between the different Semitic families of Hebrews, Arabs, and Negroes, are so marked that we can but think that persistent climatic influences have played a very large part in contributing to this result.

In *warm* climates skin and liver are particularly active. The

nervous system is exeitable and depressed alternately, aceording to the season. The abdominal organs beeome suseeptible to outside influenees. The digestion is often impaired. The indigenous inhabitants are dark-skinned and blaek-haired.

In *temperate* regions, where the best types of physieal development are to be met with, the body equilibrium is well maintained. In *cold* elimates the strain is rather on the kidneys and lungs; muscular aetivity ensures physieal development, the digestion is vigorous, and the nervous system sluggish.

Humidity has a eonsiderable effeet on the body. A relative humidity of 75 per eent. is best ealculated to suit the needs of the majority of people. If mueh in exeess of that figure, skin perspiration and evaporation from the lungs are largely eheeked, favouring the retention of an injurious amount of effete produets. Both heat and cold are badly borne when the humidity is exeessive. On the other hand, defieient humidity induees inereased evaporation with retention of urea.

Changes of temperature are often most injurious, especially when sudden. Change from eold to heat may eause diarrhœa or other intestinal eonditions, while that from heat to eold is very apt to produee eatarrhal mischief or hepatic and renal eongestion.

Winds.—A hot wind, if not too highly saturated with moisture, will inerease the body evaporation. A cold wind, in proportion to its veloeity, will extraet heat from the body. Amongst the better known tropieal winds may be mentioned the khamsin and simoon, winds of the desert, and the siroeeo of the Sahara, all dry and hot; the south-west monsoons of India, whieh blow from the middle of June to Oetober, and are hot and moist. They are followed by the north-east monsoon, which is eool and dry. Towards the south of the Malay Archipelago and north of Australia the seasonal winds are more from the north-west and south-east. In some parts of the West Indies there is a perpetual easterly trade wind, fairly dry and eool and healthy. In other parts, sueh as the larger islands, there is a daily land and sea breeze; all day the wind blows in from the sea, and soon after sunset it takes a seaward set from the land. This is exactly opposite to the eonditions of the Madras eoast, where the direetion is from the land all day and from the sea towards evening.

Influence of Climate on the Physiological Functions.

Temperature.—*Crombie's* series of extended observations on body temperatures in Caleutta have eonfirmed the law laid down by *Becher*, that the body heat inereases in the proportion of 0·05° F. for every 1° F. inerease of the air. He found that the body temperature of the European living in Bengal is about 0·41° F. higher than the average of healthy persons in England. He also found that the average temperature of the native is quite 0·5° F. higher

than that of the European, whilst, at noon, there is almost 1° F. difference between them, probably due to their large meal between 9 and 10 a.m.

The effects of food and exercise on the temperature are much the same as in temperate climates, except that exercise acts more immediately and powerfully, and the elevation of temperature may be maintained for some hours.

Sleep is found to depress the temperature in the tropics. It has been frequently said that the older resident perspires more freely than the new arrival. When this is the case it is probably due to an excessive indulgence in iced drinks, especially containing alcohol, or else to the debility caused by too long a residence without a change to a colder climate. It may be laid down as a maxim that (unless taking active exercise) excessive perspiration is only marked in the case of new comers or in old residents who need a change.

Respiration.—*Rattray*, *Francis*, and others have established the following:—

(*a*) The capacity of the chest for air is considerably greater in the tropics, the spirometric increase amounting to 7 or 8 per cent. This is due to the fact that he lungs contain less blood and have more room for air.

(*b*) The frequency of the respirations is diminished.

(*c*) The respiratory act, as a whole, is lessened. This is due to the fact that, although the chest capacity is greater, yet the diminished respirations more than counterbalance it; and, moreover, heated air contains less oxygen per cubic foot, which further reduces the total consumption.

(*d*) The elimination of CO_2 is decreased, owing to the fact that the amount thrown off bears a ratio to the quantity of air inspired.

(*e*) *Post-mortem*, the lungs of European tropical residents are lighter than the European standard.

Urine.—The humidity of the air, the amount of fluid ingested, the atmospheric temperature, and the amount of sexual indulgence, all have their influence on the amount of urine secreted. With greater humidity of the air less moisture is lost by skin and lungs, and consequently more by the kidneys and intestines. The ingestion of much fluid, especially if alcoholic, increases both the skin and kidney elimination. Sudden chills may disturb the physiological body equilibrium, and result in congestions to which the kidneys and liver are especially liable. Excessive coitus largely increases the amount of urine secreted—probably a reflex vasomotor effect on the vessels of the kidney.

Rattray calculates that nephritic vascularity and secretion are reduced by 17½ per cent. as a general average in a warm climate. The occurrence and interpretation of urea and the various salts in urine, in the tropics, have never been properly worked out. *Fowlie* has found in Singapore that phosphaturia is very common amongst Europeans, especially pregnant women, generally in the form of

triple phosphates in a slightly alkaline urine. This often has a pathological manifestation in the shape of renal colic.

Uric acid is not uncommon, but urates are rare.

Nervous System.—At first there seems to be functional exaltation lasting for some months, and followed by depression. *Birch* thinks it probable that the chemical and physiological effects of sunshine may play some part in this early exaltation, before there has been time for the depressing effects of constant heat to develop the opposite condition. The vasomotor system will share in the general depression, and the deeply-seated viscera will be liable, therefore, to congestions in cases of external chill.

Disturbance of sleep in very hot weather cannot but seriously interfere with nervous recuperation.

The tropical incidence of diseases of the nervous system, compared with other diseases, is not much higher than it is in colder climates. Taking all races into consideration, however, acute mania with secondary dementia is, in many parts of the tropics, excessive. In the Malay States, for example, such cases often constitute over 50 per cent. of the admissions under diseases of the nervous system.

Menstruation.—The advent of menstruation is largely influenced by tropical climates.

The following table is based on nearly 1500 observations by *Das* in Calcutta, compared with *Madden's* English observations.

Thus, generally speaking, the average English girl first menstruates at 15, but if born in the tropics, or brought up under tropical influences, it will be at 14; and the Eurasian girl at 13 years of age. On the other hand, the average age for the native girl is 11.

As *Barnes* has said, "Certain races preserve the menstrual type proper to them in the country of their origin, even when transplanted. Jewesses, whatever be their habitat, menstruate generally somewhat earlier than girls of Saxon origin."

Birch states that apparently abortions and miscarriages are more common amongst Europeans in India than in their native countries; and European women at the climacteric are unduly liable to hæmorrhages.

Acclimatisation.

Acclimatisation is that process by which animals or plants become physiologically adapted to a climate different from that in which they are indigenous.

There is a distinct effort on the part of nature to accommodate the organism to a new environment, and the amount of its successful effort in this direction represents the limit of possible acclimatisation. This effort of nature is a very complex one, and, as far as man is concerned, probably means the gradual adaptation of the body to the new circulatory distribution, together with a continuous pressure

Class.	Percentage who Establish Menstruation at the following Ages:—								
	10	11	12	13	14	15	16	17	18
English girls in England,	...	...	3·6	10·4	19·6	**28·8**	21·9	13·6	2·1
European girls under Indian climatic influences,	·9	6·9	12·9	18·2	**30·2**	12·9	12·9	4·2	·9
Eurasian girls in India,	·48	5·5	18·6	**28·0**	26·8	14·8	4·07	·95	·72
Hindu and Mohammed Natives of India (1·11 at 9 years of age),	8·29	**33·15**	30·36	16·13	6·27	3·81	·33	·22	·11

brought to bear upon the various physiological functions dictating the acquirement of general hygienic habits suited to the new state of affairs.

Certain eminent writers have denied the possibility of acclimatisation, but the evidence with regard to man is so overwhelming, that it is as impossible to deny it as to deny the acclimatisation of other animals and of plants which are matters of every day knowledge and observation.

Almost all the domestic animals of Europe, now so well acclimatised to temperate and cold climates, were originally indigenous to the tropics.

"If the human race constitutes a single species, then the mere fact that man now inhabits every region, and is in each case constitutionally adapted to the climate, proves that acclimatisation has occurred. But we have the same phenomenon in single varieties of man, such as the American, who inhabits alike the frozen wastes of Hudson's Bay and Terra del Fuego and the hot, low, equatorial valleys of the Andes. No doubt a sudden transference to an extreme climate is often prejudicial to man, as it is to most animals and plants; but there is every reason to believe that, if the migration occurs step by step, man can be acclimatised to almost any part of the earth's surface in comparatively few generations" (*Wallace*).

"The best examples of acclimatisation are found where European races have permanently settled in the tropics, and have maintained themselves through several generations. Two sources of fallacy have to be guarded against:—(1) The possibility of a mixture of native blood, and (2) a succession of immigrants from the parent country intermingling with the colonists. These are reduced to a minimum in the cases of the Jews, the Dutch in South Africa, and the Spaniards in South America; all marked instances of an acclimatising faculty" (*Davies*).

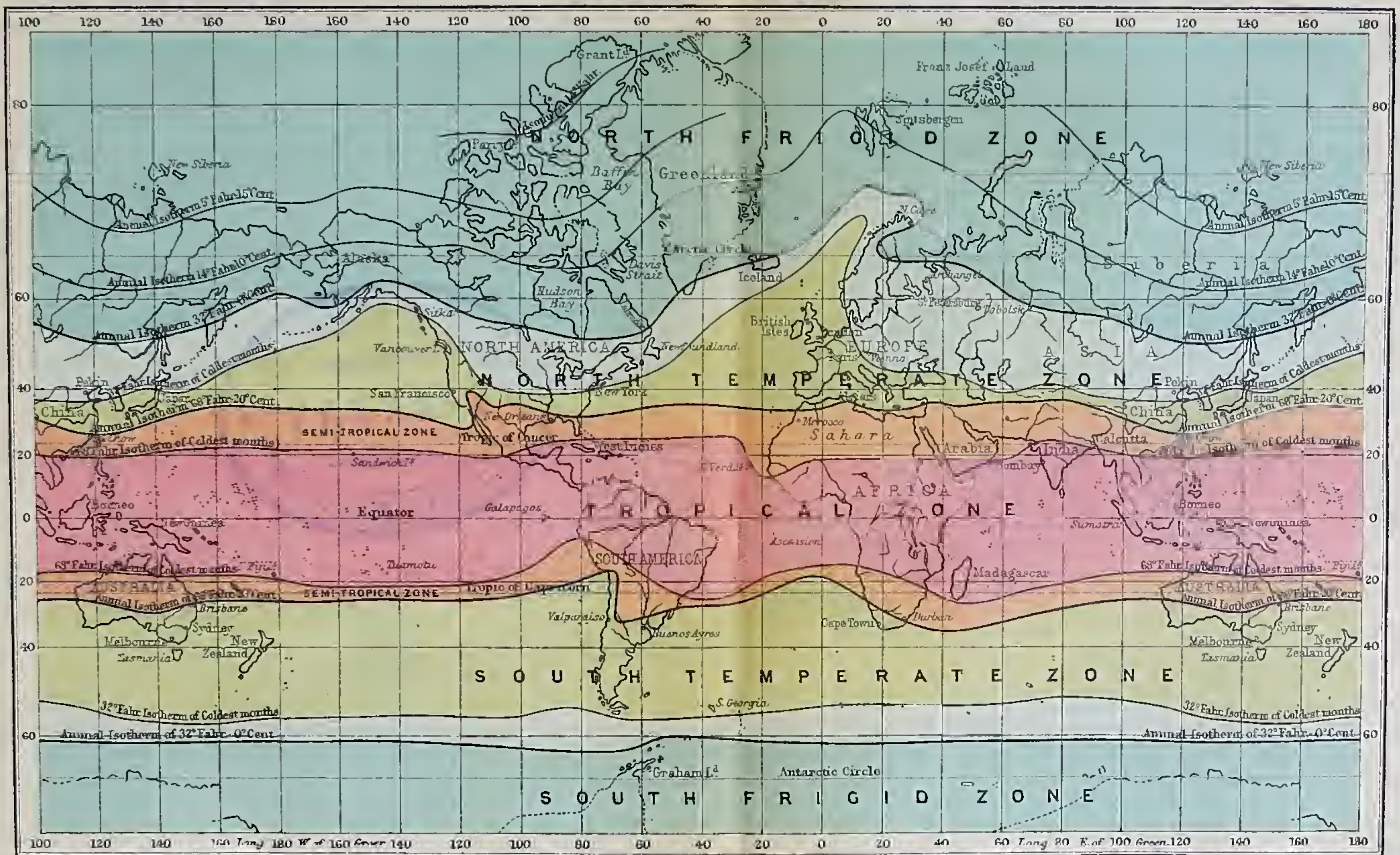
According to *Arnould*, a race is acclimatised when it preserves:—

1. Its force of demographic expansion (*i.e.*, the natural increase of population).
2. Its normal longveity.
3. Its aptitude for physical and intellectual work.

According to *Bertillon*, the following influences tend to prevent rapid acclimatisation in new isothermal regions:—

1. Acute diseases, endemic or epidemic.
2. Chronic anæmias diminishing powers of disease resistance.
3. Prevalence of infant disease amongst the offspring of the newcomers.
4. Physical and intellectual degeneration and infertility of second and third generations.

CLIMATE CHART



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Arnould gives the following *conditions favourable to the process of* *cclimatisation* :—

1. *Slight alteration in latitude*, if possible from a warmer to a older climate.

E.g., English and French colonists in Canada, and by opposite conditions the Germans at San Leopoldo in Brazil, where 120 families number 120,000 persons after 45 years.

2. *Ethnical disposition.*

E.g., Jews are ubiquitous without deterioration or loss of individuality. The Chinese, who thrive everywhere—in Malaya, India, N. America, and Australia. They are a temperate, thrifty, and energetic race. The Spanish and Portuguese have thriven well in S. America.

3. *Manners and Customs of the Colonists.*—Diet, clothing, and eneral habits should be adapted to the climate, as practised by ews, Chinese, Spaniards, &c.

4. *Aptitude for Cross-breeding.*—The product of the Spaniards ith the American Indians is a very different and more permanent ce than the half-breed of the Anglo-Saxon and Indian in North merica.

5. *Soil and Locality.*—A dry, healthy, and productive soil in a ountry not overcrowded and peopled by slow emigration is conicive to increase of fertility and speedy acclimatisation.

Climate Zones of the World.

North frigid zone,	mean annual	temperature,	20°	Fahrenheit.
North temperate zone,	,,	,,	50°	,,
Tropical zone,	,,	,,	80°	,,
South temperate zone,	,,	,,	50°	,,
South frigid zone,	,,	,,	20°	,,

Average Rainfall at Different Latitudes.

Latitude.	Inches.	Latitude.	Inches.
0° Equator	100	50°	30
20°	80	60°	20
30°	60	70°	10
40°	40	80°	5

Temperature and Rainfall of Certain Cities.

Cities.	Latitude.	Mean Annual Temperature.	Mean Summer Temperature.	Mean Winter Temperature.	Mean Annual Rainfall.
London, . . .	51° 32′ N	50·1	63·8	37·3	24
New York, . .	41° 6′ N	51·7	72·3	31·4	36
Pekin, . . .	39° 53′ N	54·8	81·1	26·7	28
Calcutta, . .	22° 36′ N	79·5	86·7	72·2	70
Hong Kong, .	22° 17′ N	76·5	84	75	73
Bombay, . .	18° 57′ N	81·2	82·8	77·4	80
Kingston, Ja., .	18° N	78·1	81	77	34
Colombo, . .	6° N	81	85	80	78
Singapore, . .	1° 15′ N	80·8	83	79·4	97
Sydney, . . .	33° 54′ S	62·7	69·6	54	52
Cape Town, .	34° 56′ S	64·7	70	58·3	24
Melbourne, . .	37° 49′ S	57·6	65·2	49	31

CHAPTER III.

FOOD, EXERCISE, CLOTHING.

Food.—The ingestion of foodstuffs is, of course, necessary for the naintenance and repair of the body tissues in which the proteids nd salts take a large share. The second large class of foodstuffs—ne carbohydrates and the fats—are largely concerned with the roduction of energy exhibited as either heat production or nuscular work.

In the oxidation of carbohydrates only sufficient oxygen is equired to oxidise the carbon, as the hydrogen and oxygen already xist in the proper proportions to form water; in fats, however, dditional oxygen is required to secure the oxidation of the ydrogen as well as of the carbon.

In cold climates this oxidising power of the carbohydrates and ats is utilised to secure heat production.

In cold climates, moreover, the muscular activity which is ndulged in to secure warmth, is instrumental in utilising the noxidised products of metabolism which result from an excess of roteids in the diet.

The new comer to the tropics is too often ignorant of, or fails grasp, the importance of these considerations.

He adopts a diet very similar to that to which he has been ccustomed at home, say three meat meals a day, sweets twice, entiful bacon, butter, &c., and, in addition, very likely a surfeit the starchy banana.

The heat makes him disinclined for much exercise, and leads the consumption of a far greater amount of liquid than is either ecessary or desirable.

The result is obvious. The surfeit of proteids, without sufficient odily exercise, causes a rapid metabolism, resulting in a loading the blood with unoxidised products, an excessive output of ic acid, a liability to the formation of boils and abscesses, and equent disorders of the intestinal tract.

The excess of the non-nitrogenous material in the diet gives rise fat production, biliousness, dyspepsia, and an over-production heat in climates where the heat production should be reduced a minimum.

Salts are an essential of diet in tropical as in temperate climates.

A considerable amount of sodium chloride may be taken with vantage in warm climates—a fact recognised by many natives.

The carbonate forming salts, such as the tartrates and citrates occurring in fruits and vegetables, with their anti-scorbutic principles, are essential for maintaining the alkalinity of the blood.

Passing from the theoretical to the practical we may briefly consider the circumstances and necessities of tropical food.

Milk.—Goat's milk and buffalo milk are not uncommonly used in the East. The former is richer in cream and poorer in proteids than cow's milk. Moreover goats are not clean eaters, and it is as well to be careful in making use of their milk; buffalo milk contains from 7 to 9 per cent. of fat (double that of cow's milk), a circumstance of which the wily native milk-seller is well aware, and makes use of the nearest watercourse to his own advantage.

It is necessary to be very careful in selecting cow's milk, for the owner, to save money, will often, in India, feed his stock with stable litter, or turn them loose in the streets to pick up what they can. The author has actually seen cows in the West Indies eating the family washing, which had been left out to dry on the bushes of an open compound. The milk from such cows is naturally not worth the high price which is frequently demanded.

Meat.—As a rule, live stock is not very well nourished in the tropics. The meat is often infected with cysticerci of various kinds, and should therefore be well cooked before being eaten.

Vegetables.—Raw vegetables are especially apt to be contaminated in the tropics. The dirty personal habits of natives, the custom of watering vegetable gardens with night-soil, the contaminated dust blowing about the streets of an eastern or western town, the habit of bringing vegetables from China as a deck cargo in Coolie ships where the Coolies endeavour to keep them fresh by micturating on them—all are urgent indications for avoidance, or extremely careful treatment, in those cases in which the vegetable is not to be cooked.

Water.—Water in India is largely taken from canals and shallow wells. These, of course, are liable to grave suspicion, owing to the native habit of washing clothes and person in the nearest water source.

Wells are frequently the only source of water in other tropical places. On the other hand, catchment areas and tanks are sometimes utilised to furnish the water supply.

In most of the larger tropical towns, however, there is at the present time some catchment reservoir with a proper main delivery and filter beds. Water from such a source is generally fairly reliable.

As a general rule, the safest plan is always to boil and filter any water intended for drinking purposes. The Pasteur-Chamberland filter has perhaps proved the most reliable, being strong, portable, and easily cleaned.

The drip-stone filter so often seen in semi-civilised native regions has but little use except to separate mechanically any gross pollution.

Alcohol.—The physiological effects of alcohol are fairly well known.

It dilates the cutaneous vessels, and lessens body heat by radiation. It first excites and then depresses the heart.

In small quantities mental activity is stimulated by the increased quantity of blood sent to the brain; larger quantities, however, paralyse and depress the nerve centres.

In moderate doses it induces sleep and assists digestion.

While not absolutely necessary for a man in any climate, a small amount, well diluted, is often beneficial in the tropics.

It should *never* be taken (unless with the mid-day meal) before sundown. A certain amount at that time has some effect in checking waste and in removing fatigue incidental to the strain of the day's work.

The best form in which to take it is that of a light claret diluted with water or soda, or else in the form of a pure whisky well diluted. Three ounces of whisky in twenty-four hours is the maximum which should be taken by a man in health.

Beer, except the lightest lagers, is not a sound drink for the tropics. Excess of alcohol is added to it for "keeping" purposes; the contained lupulin is depressing; oxidation is lessened, with a consequent liability to a deposition of fat.

The excessive use of alcohol is a predisposing cause of hepatitis, dysentery, diathermasia, and phœbism.

Other Beverages.—Tea, coffee, and cocoa are all excellent beverages for tropical climates. The contained alkaloid is a valuable stimulant to the nervous system.

Lime-juice is valuable as a thirst quencher and antiscorbutic. Taken with barley water it is an admirable drink for women during the day time, although it should be replaced by some form of alcohol at the evening meal.

Excess of condiments or highly-spiced curries, &c., should on no account be indulged in.

A final remark on *Tinned Foods in the Tropics* might well be made here.

Daniels recalls that the late Dr. H. E. Mann, in March, 1902, advocated some action being taken to compel manufacturers to stamp their tins with the date on which the food is prepared.

In some parts of the tropics, tinned food looms large on the dietetic horizon of the European, and, therefore, some such method of restricting abuses is urgently required.

The life-history of a tin of meat is, in many cases, as follows:—

"The tin is imported by a reputable firm in the first instance, but every year or so they have a sale of surplus stock which includes many tins of meat which have been kept for some time. These are bought up by up-country store-keepers or small retail dealers. Any tins that are 'blown'—that is, in which sufficient decomposition has taken place for the gas formed to cause the end of the tin to bulge—are punctured, reboiled, and the puncture covered with a drop of solder. This process can be repeated."

Exercise.—A certain amount of regular exercise is a necessity in tropical climates.

This statement may appear somewhat of a platitude, but it can be better appreciated after some years of tropical residence. The temptation to lead a sedentary life is often great. If fought against by rational and moderate exercise, the best conditions are secured to maintain a healthy life.

Exercise aids digestion; stimulates the liver, and intestinal peristalsis—thus avoiding the oft-told tale of constipation. It relieves the body of any excess of CO_2 by increasing the output, and thereby prevents undue irritation of nerve centres already weakened by prolonged residence in the tropics.

The actual amount of necessary exercise and rest will vary with each individual.

The exercise should not be overdone, and should be taken in the early morning or late afternoon.

Riding, where available, is of valuable use in hepatic congestion and constipation.

A round of *golf*, or two sets of *lawn tennis* will prove suitable exercise in many cases.

Bicycling in moderation may be indulged in, but hill-climbing should be avoided.

For those in health, *shooting* is an admirable sport—it secures exercise and relieves the monotony of the daily routine. When obtainable *sea bathing* is highly beneficial to many persons.

If it is found necessary to sit about after taking active exercise, a warm sweater should be worn under the coat, and also a scarf round the neck, since a perceptible fall of atmospheric temperature usually occurs during the short tropical twilight.

A *bath* may be taken with advantage twice daily in the tropics. Unless taken from deep wells, it is seldom too cold to be used by those in health.

A preliminary soaping with some unirritating soap, followed by a cold water sponging, is perhaps the best method.

It is highly important, however, that old malarial cases, dysenteric cases, those with renal disorders, hepatic congestions or obesity, and people of advanced age should never use a cold bath in warm countries. It should be either tepid, or, preferably, warm.

In warm climates the nervous system demands more *rest* than in temperate ones.

At least eight hours' sleep should be secured by adult males, and nine hours by females. In excessively hot situations—such as the plains of India during the hot season—part of this sleep should be obtained in the middle of the day.

An interval, however, should elapse after taking a meal, otherwise the sleep will be heavy and deep, and perspiration profuse.

In cases in which excessive work, either physical or mental, has to be done, ten hours' sleep is none too much.

Occasionally to rest quietly in a long chair for an hour or so doing

nothing is a practice which might be more advocated, and with advantage indulged in.

Sleeplessness is often the first sign of a breakdown in health. Such cases should be carefully enquired into, and, if not amenable to rational treatment, may indicate the necessity for furlough.

Bedrooms should be lofty and well ventilated, and should contain a minimum of furniture. The mosquito-net in common use is a snare and delusion. It usually is affixed in a limited box-like shape, closely surrounding the sleeper, the top being closed in with a sheet of linen or cotton at a height of about 6 feet. The mesh of the net hinders a proper diffusion of air, while the vitiated atmosphere within, which cannot escape at the top, gradually envelopes the occupant, who wonders in the morning what can be the reason for his dreams and heaviness. The better plan is either to have a permanent mosquito-room—or else a very large net suspended on a frame from the ceiling—within which there is ample room for beds, table, &c.

Clothing.—The subject of suitable clothing for Europeans in the tropics has never been adequately dealt with in any text-book of medicine or guide to travellers.

The argument is always based on faulty premises.

The tune that is harped on is **HEAT**, with two strings—"woollen underclothing" and "white outerclothing."

This is entirely opposed to the lessons taught by nature and observation.

What has nature done for the native, who for centuries has inhabited the tropics? A visitor from Mars, after perusal of articles on tropical clothing, would at once conclude that she had furnished him with a pure white skin. Now, we all know that the very opposite is the case; the skin is invariably black or brown. Why? Because the actual heat of the sun (solar radiation of red heat-rays) is seldom sufficient to be injurious to any human beings except those whose thermo-taxic mechanism has been shattered by alcohol or overwork.

The highest recorded solar radiation temperature (*i.e.*, the red "heat-rays" of the spectrum) is 175° F., and that is rare, the mean being 130° to 160° F. Healthy men in stokeholds, glass factories, and elsewhere can and do bear such temperatures every day with impunity. The difficulty which we have to face, therefore, in the tropics is not the heat but the **LIGHT**. They are the blue, violet, and ultra-violet light rays of the spectrum, which give us sun-erythema, fever, and phœbism, and it is against these that nature has vainly tried to teach us the lesson of the native skin, which we daily preach and act against.

Let us take the second string, and see what nature would teach us.

What does the native do in the hottest climates? He clothes himself as scantily as possible. When civilisation demands it he wears cotton (not because it may be white, but because it is

light), and is far more comfortable than the European who wears flannel.

If sheep are transported to a hot climate they gradually lose their thick imbricated wool, which prevents proper evaporation from the skin, and smooth hair grows in its place.

Lions and other animals which are furnished with thick fur in colder regions have only thin hair in the tropics.

Wherever we turn, therefore, we see that nature adapts herself to her surroundings. The European, however, who wears flannel to keep himself warm in cold climates wears it also in hot climates! Nothing could be less reasonable. It makes him hotter; the imbricated scales irritate beyond patience, and are a fruitful source of prickly heat; the material absorbs moisture very badly, and the excessive perspiration is kept in close contact with the body; and this saturated envelope of moist air checks the further action of the skin in a dangerous way.

The following are the usual clothing materials:—

I. *Of Vegetable Origin* (cotton, flax, jute, &c).—These materials, especially the first, are the most suitable of all for underwear in the tropics. They are thin and very light. The fibres are smooth and unirritating; they absorb water quickly and part with it quickly, thus allowing an efficient and constant action of the skin. The speedy evaporation does not cause undue contraction of the skin capillaries since outer-garments are worn as well as under-garments.

The point to be observed is—that, owing to their excellent absorption of waste skin products, it is necessary to change at least twice daily.

(*a*) *Cotton.*—This is the down surrounding the fruit of a *Gossypium*—a tropical shrub cultivated (for textile use) chiefly in the southern part of the United States of America, Brazil, India, and Egypt.

It is manufactured into **CALICO, CRAPE, SATEEN, FLANNELETTE, DRILL.** The two former are suitable for underwear. A material called "Japanese crape" being the most suitable of all for loose shirting to be worn next to the skin.

(*b*) *Flax.*—This is the fibre of a plant called *Linum usitatissimum* or "Linseed"—a small annual, about 2 feet high, with pale blue flowers. It grows in the tropics, sub-tropics, and temperate climates: and its economic use is of great antiquity.

It is chiefly manufactured into **LINEN, DIAPER** (= d' Ypres—a town in Flanders celebrated for the manufacture of the finest table linen), **SEWING-THREAD, TOW, CAMBRIC, HOLLAND, DRILL.** It is woven in much the same way as cotton. It is extremely absorbent of moisture, though less so than cotton.

A fine cambric is a cool, durable, light, and efficient material for underclothing.

(*c*) *Jute* is a fibre from the *Corchorus capsularis*, a plant chiefly cultivated in Bengal. It is somewhat coarse, and is used both for

the manufacture of native clothing and for the adulteration of fabrics at home.

II. *Of Animal Origin* (leather, silk, wool, &c.).—Speaking generally, these materials are of much less value than vegetable fibre, as underwear for tropical use.

Wool is especially valuable in *cold climates*. The rough imbricated scales are a valuable stimulus to cutaneous circulation. Owing to scanty perspiration the non-absorbent qualities of the wool do not prove a bar to its use. The minimum of cutaneous excretion in cold climates avoids the fouling of its coarse texture which occurs invariably in the tropics. While the considerable air interstices form a buffer for retaining warmth and excluding cold.

(*a*) *Leather*, or the tanned skin of animals, is used for several clothing purposes:—Boots, shoes, belts, gloves.

For boots and shoes, leather is not as good a material as is canvas for tropical use. Canvas allows of freer ventilation for the feet, which is a great point to be secured. It is, moreover, sufficiently comfortable, yielding, and of neat appearance.

(*b*) *Silk* is the fibre produced by the *Bombyx mori* or silkworm in China, Japan, India, Italy, and France.

Either alone or as a mixture, it is a common element in clothing materials. Piece silk, velvet, satin, ribbon, gloves, stockings, &c., being amongst the number. As an article of tropical underwear silk is superior to wool, but much inferior to cotton or flax. It absorbs moisture more rapidly than wool, but much less rapidly than the vegetable fibres.

It is very expensive. It is smooth and soft and has not the same deleterious effect as wool in retarding efficient skin action.

It is an ideal underwear for the sub-tropics or for the warmer seasons of temperate climates, but inferior to cotton for the tropics.

(*c*) *Wool*.—A form of cutaneous hair in animals. For purposes of clothing this is made up either alone, or more frequently mixed with cotton.

It is an especially bad material for tropical underwear, and this for many reasons. First, the fibres have imbricated scales which are irritating to the skin, and a frequent source of prickly heat.

It does not absorb moisture quickly, thus hindering the normal output of perspiration. Being loosely woven, it holds much air in its interstices, and thus the outside atmosphere can with difficulty penetrate it, and the same layer of warm moist air remains in contact with the body—checking the proper action of the skin and increasing the feeling of heat.

Note.—It is a great mistake, made by Jaeger and others, to imagine that wool is "porous" in the sense of allowing a free passage of air through the material. It is for the very reason that it is *non-porous* that it forms such a valuable clothing material for cold climates; the large amount of contained air keeps the body surface warm, and prevents both skin evaporation and the entry of air from the outside. Diffusion is sufficiently slow to be neglected.

Lastly, the coarseness of its surface and thickness of its texture (compared to cotton) favour the retention of the skin waste-products, necessitating just as frequent and much more careful washing than either cotton or linen.

In Jaeger's garments the long staple wool fibres are used without any admixture of cotton; and undyed.

In the form of a loose shirt it is suitable for a day's shoot, but the closely fitting under-garments should never be used by those who value their health or comfort.

External Clothing.—We have already seen that the noxious element in sunlight is the violet and ultra-violet actinic light, and that nature has made the native skin and hair into a colour-screen opaque to these rays.

Habit and custom, however, have more or less ordained throughout the tropics that white is the colour to be worn. This is usually in the form of white drill, and is unsuitable for many reasons. In the first place, thin white material, such as this, is almost transparent to the actinic rays of the tropical sun, and it does not therefore serve to protect us from this dangerous element. *Secondly*, while the white surface certainly reflects many of the longer red heat-rays, yet the material is too thin a texture to totally exclude them, and, consequently, it is by no means as cool as we could wish when actually in the sun. *Thirdly*, when the thin material gets wet from excessive perspiration, its texture is such, that rapid evaporation takes place, and the body is too rapidly chilled. To obviate this some form of woollen undergarment has to be worn, and prickly heat, deranged skin action, &c., supervene. *Fourthly*, the reflected white light is excessively annoying (and in course of time injurious) both to the eyes of the wearer and to those in his neighbourhood. The amount of light thus distributed is perhaps hardly realised. Let the reader but sit in a darkened tropical room with an open door at noon, and observe the extra light which momentarily fills the room as a person in white garments passes by in the bright sunshine, even at a distance of 20 to 25 yards. In the tropics, nature clothes the world in evergreen garments and man in black; man, thinking himself the wiser judge, decks himself in white, and persists in white-washing or white-painting every available surface.

Valuable researches have been made by *Sambon* and *Baly* on this question.

Spectroscopic investigations on native skins has shown that the pigment has a strong protective effect against the passage of the injurious actinic rays.

With the object of obtaining some material which should exclude the actinic rays, and reflect the heat rays, *Sambon* endeavoured to produce an ideal thin cloth fabric by the interweaving of various coloured threads which should give a warp surface of one colour and a weft of another. This resulted, after many trials, in the production, by a large firm of manufacturers, of a material named as *Solaro* fabric.

The beautiful white reflection was overcome by the use of a khaki colour, which is practically as efficacious in reflecting the long rays as is white, and the material was found by *Baly* to be as impervious spectroscopically to the actinic rays as is the native skin in tropical countries.

The method of manufacture is to use threads of yellow and blue, each twisted separately and then together, for the warp, and red threads for the weft, bringing the weft threads to the back in the proportion of three to one—thus giving the front a proportion of three yellow, three blue, and one red, which secures a perfect khaki effect on the outer surface and a red colour screen on the inner surface.

Such material is ideal for tropical use.

Thin blue serge is another material not to be despised. European tailors have yet to learn, however, how to produce light-weight garments for tropical use.

The coat should preferably be double-breasted. Such a coat the author possesses, weighing only *14 ozs.*, and trousers of the same material, weighing *10 ozs.* Such attire is quite suitable for ordinary daily use (other than during active exercise), and all that is needed to complete the attire is a Japanese crape shirt with starched cuffs and turned-down collar, weight 7 ozs.; a pair of socks, weight ½ oz.; canvas shoes, weight 12 ozs.; and a Manila straw wide-awake, lined with red silk, weight ½ oz. Tie, negligible.

Total weight of complete outdoor outfit, 44 ozs. (or less than 3 lbs.)

It may be of interest to note that a blue serge coat made by an English tailor, as the lightest possible material and make, for the tropics, weighed 30 ozs.

We now come to the question of **headgear.**

The following essentials of a rational headgear should be secured:—

(*a*) The lining should be of red or orange silk, preferably one thickness of each, to cut off the actinic rays.

(*b*) The hat should be of light weight, not more than 5 ozs.

(*c*) The brim should slope downwards, and its edge should project horizontally to a distance of at least 4 inches from the wearer's head throughout the whole circumference.

(*d*) There should be some means of ventilating the interior either by an apical ventilator, or by openings between the head band and the hat.

(*e*) The external surface should be a good reflector of long rays, or else the material should be non-diathermanous, since the internal temperature should be lower by *at least* 25° F. than the mean solar radiation.*

The following data of a few ordinary tropical hats will show where shortcomings exist. The hats themselves are depicted in Fig. 3:—

* Deduced by the author from a series of experiments on the internal temperature of hats exposed to tropical noonday sun.

Hat.	Weight, in ozs.	Brim, in inches.
1.* Khaki service solah topi,† . .	$14\frac{1}{2}$	Back, . 4 Front and sides, . 2
2.* Ordinary type of eivilian topi (Ellwood's patent), . .	10	All round, 3
3.* Bangkok grass topi, . . .	3	All round, $4\frac{1}{2}$
4. Ordinary Panama hat, . .	2	All round (if turned down), . $2\frac{1}{2}$
5. Manila-straw wide-awake, .	$\frac{1}{2}$	All round, 3

An inspection of these details will show that the service topi has not got a suffieiently protective brim, and is, moreover, about three times too heavy.

In faet, it is almost as eriminal to expect a man to carry about that weight as to make him wear an overcoat under the skies of brass.

The eivilian topi depicted is double the proper weight and should therefore at once be eondemned, although otherwise it fulfils requirements.

The Bangkok grass topi, when properly lined and made up, is as near perfeetion, for the most extreme heat of the tropics, as a hat eould be.

The last two, especially the most excellent Manila straw, are admirable for early morning or late afternoon wear.

Women's Clothing for the Tropics.—The practitioner may be called on to advise in this matter.

In those regions of the tropies in which there are distinct cool and hot seasons, some modifieations will be necessary.

In the hot weather the best material for underwear is fine, soft cambric—ehanged twice or thrice daily—which will obviate much priekly heat. It is best worn in the form of a chemise and drawers, since thus there will be two folds ovei the abdomen besides the superimposed garments. In the cool season, eombinations of thin soft natural wool are the most suitable underwear. The stays should be of open cellular material, of a coffee-colour, whalebone frame. Thin eashmere is the best material for stoekings.

* Ventilated.
† *Not* derived from *sol, solis*—the sun.

[*To face p.* [illegible].]

Fig. [illegible]. Types of Tropical Headgear.

Civilian topi
(weight, 10 ozs.).

Military service topi
(weight, 14½ ozs.).

Bangkok grass topi
(weight, 3 ozs.).

Manila-straw wide-awake
(weight, ½ oz.).

[*Photo by Author.*]

The foot is normally slightly larger in hot climates, and boots or shoes should never be too tight.

Petticoats and dresses may be as fashion dictates.

The usual desire for soft white hands is best attained by the use of the thinnest French kid gloves. Cotton and silk gloves are transparent to the ultra-violet rays which cause sunburn. Suede gloves are too hot.

A solah topi is not a necessity for women if a suitable parasol is used.

The hats worn should always be of a light weight and lined inside with red silk.

The parasol should not be too flimsy. A thick red or orange material should be the basis; and it could be covered with lace and lined with chiffon, or otherwise, as fancy may dictate or fashion demand.

CHAPTER IV.

HYGIENE OF THE MOUTH.

The care of the mouth, always a matter of importance, owing to its share in the maintenance of health, becomes even more so in the tropics where fermentative changes are especially prevalent, and where digestive disorders are apt, with the smallest encouragement, to assume grave proportions.

Salivary calculus (tartar) is perhaps more common in warmer than in colder climates.

Pyorrhœa alveolaris is likewise widespread, and its preliminary stage—receding gums—often proves extremely difficult to deal with.

A few notes on these subjects and on dental caries will not, therefore, be out of place.

Salivary Calculus.—Salivary calculus, or *tartar*, is a cretaceous substance (composed of phosphate of lime, with epithelium, debris of food, &c.) which has a tendency to deposit on the teeth, especially at the back of the lower incisors and on the buccal surface of the upper molars. When rapidly formed it is comparatively soft and readily broken, but when more slowly deposited it is very hard and difficult to remove.

The lime salts are normally present in saliva in a state of solution.

Tartar deposited from the parotid gland is usually of the soft variety, while that from the submaxillary and sublingual glands is hard.

An analysis of tartar has been found to yield :—

Earthy phosphates, ...	79·0
Salivary mucus, ...	12·5
Ptyalin, ...	1·0
Animal matter, ...	7·5
	100·0

Tartar is normally white or pale yellow in colour, but may be stained brown, green, or black from decomposition in the mouth, or from the use of tobacco, port wine, or other colouring matter.

Occasionally tartar is found deposited in narrow, hard, dark rings, just beneath the free margin of the gum ; it may occur on any teeth, but is most commonly found encircling the necks of the upper incisors.

A further variety is found as hard dark granules, often extending a long way up the root of a tooth, even to its apex, and is then frequently associated with so-called Rigg's disease.

Loose teeth are frequently encased in a complete sheath of tartar, and in some cases this acts as a splint, binding the teeth firmly together, and affording them considerable support. Salivary calculus is also found to deposit on artificial plates which are not duly cleansed.

When tartar is deposited in considerable thickness, it assumes a wedge shape with the base towards the gum, on which it acts as an irritant, causing its gradual shrinkage from the neck of the tooth, the alveolus also being absorbed before the advancing irritation; the root of a tooth may thus be laid bare almost to its apex, causing death of the pulp from cutting off its blood supply. If the tartar is carefully removed, the gum beneath will be seen congested and covered with an abnormally thin layer of epithelium, so that bleeding is readily produced.

Tartar is deposited in large masses as the result of the disuse of certain teeth, as where—in consequence of an exposed pulp—mastication is carried on entirely on the opposite side, or when, from the loss of teeth, the remainder have no antagonists.

Dr. Miller, writing on this subject, states that "Normal saliva contains calcium phosphate as well as carbonate; these are held in solution in the blood and in the glands by carbonic acid. When the saliva enters the mouth the carbonic acid escapes, and the lime salts are precipitated."

Treatment consists in the thorough removal of the salivary deposit by means of scalers, care being taken to injure the gum as little as possible. To prevent impeding by occurrence of hæmorrhage, the treatment should be carried on from before backwards.

After all traces of tartar have been removed the teeth should be thoroughly polished with fine powder, so as to leave no spot as a nucleus on which the deposit may recommence; and the patient should be directed to be most assiduous in the use of his tooth brush, night and morning, and to use it up and down, and not across the teeth, as it is generally employed. A tooth brush known as the "Prophylactic" is well suited for reaching the dental interspaces.

If the gums are very congested, it is useful to rub powdered tannin or some other astringent on them.

Pyorrhœa alveolaris.—*Pyorrhœa alveolaris, false scurvy*, or *Riggs' disease* is a condition in which the gum in the neighbourhood of one or more teeth is chronically inflamed, deeply congested, and readily bleeds: is rather tender to the touch, and has a slight, thick creamy discharge exuding from the alveolar socket, which may be recognised by gentle upward pressure of the finger on the gums.

Pockets form around the exposed necks of the teeth, in which food, &c., collects and decomposes. The condition has a tendency to spread, and may involve much of the dental arch.

Nodules of hard tartar form within the margin of the gum; as

the disease advances the gum is peeled off and the alveolus is absorbed, so that the tooth becomes loose, and eventually falls out.

The absorption of the alveolus takes place in a characteristic manner, a saucer-shaped hollow being formed around the affected tooth, while, at the same time, a fresh deposit of bone takes place on the outer or inner edge of the alveolus, causing a ridge which may be readily felt by the finger.

Etiology.—Various, but not wholly satisfactory, causes have been assigned for the disease.

Riggs considers it to be a bony caries. This, however, is contra-indicated by the disappearance of the disease after removal of the affected teeth.

Others consider the condition as an outcome of the tartar deposit. This is negatived by the fact that $\frac{1}{8}$ to $\frac{1}{4}$ inch of clean, smooth tooth is generally present between the tartar and the bone.

Some consider it to be a constitutional disease of middle or later life, but it is found to affect the mouths of people otherwise in good health.

A bacterial origin is probably nearer the truth. *Goadby* makes the following remarks:—

"So far my own experiments are very much in a line with those of Miller. I have isolated a large number of different bacteria, some of them pathogenic for animals, but so far no organism appears with sufficient frequency to associate it especially with the disease. The results of some inoculation experiments, however, throw some additional light upon the subject. Guinea-pigs succumbed when inoculated with the filtrate of old froth cultivations, made from the mouth direct, containing various fine bacilli (0·5 μ in width, exhibiting irregularly banded marking, and attaining thread-like dimensions of some length), and, moreover, giving off a considerable fæcal smell. No organisms were found in the tissues post-mortem, and it seems reasonable to suppose, therefore, that the animal died from a toxæmia. Such a circumstance appears to point to a toxic element in pyorrhœa, and we may call to mind the curious shining atrophic appearance of the gums in cases of long standing. What appears, therefore, to be a reasonable supposition is that the peculiar bacteria concerned in the process produce some sort of toxine, which so alters the vitality of the tissues surrounding the teeth that any and every mouth organism may assist in the continuation of the process."

Treatment.—The treatment can scarcely be considered satisfactory, for relapses are common, though the disease may be brought into abeyance.

Every trace of tartar should be removed from the gum pockets, and sulphate of copper packed in them every day for ten days, the freshly-deposited tartar being on each occasion removed before re-applying the drug.

This $CuSO_4$ is a strong astringent, and, next to the salts of mercury, is one of the best germicides. Its disadvantages are

that it dissolves but slowly, thus keeping up the exceedingly disagreeable taste, and causing many patients to fight shy of the treatment.

A compound of acid carbol 25 parts, with camphor 75 parts, may be used with advantage, especially in the later stages, the carbolic acid acting as a disinfectant, and the camphor serving to dry up the gums.

Riggs, in addition to removal of tartar, advocates chipping away of the alveolar edge, a procedure which is scarcely warranted if the condition is other than a primary osseous caries.

Of *internal remedies*, calcium iodide, gr. iij b.d.s., might with advantage be tried in all cases.

If the disease is complicated by syphilitic ulceration of the gums, they should be scraped, and iodide of potassium administered.

Dental Caries.—Dental caries, or decay of teeth, signifies the disintegration of enamel, dentine, and cementum, and is brought about by the action of various organic acids (mainly lactic) produced from the carbohydrate constituents of a normal diet, by the vital activity of bacteria.

Digestive ferments are subsequently produced by these organisms, and result in the dissolution of the decalcified matrix of the cementum and dentine.

Miller has demonstrated that artificial caries can be reproduced in tubes of dentine exposed in carbohydrate solutions to the action of micro-organisms of the acid-producing class.

Caries of enamel cannot be reproduced artificially with the same ease, as uniform denudation of the enamel generally takes place; since, therefore, natural caries takes place at one point rather than over the whole tooth, the initiation of the process is a matter of some interest.

Many theories have been advanced to explain the commencement of enamel destruction—*e.g.*, ill-developed conditions of the enamel structure, deficiency of lime salts (associated with the condition of water supply), common constituents of a diet, &c.—but none of these can be looked on as anything but pre-disposing causes.

The researches of *Miller* and *Leon Williams* have demonstrated a film-like layer of bacteria covering the enamel in sheltered positions. The acids produced by the bacteria eat out hollow spaces between the enamel prisms, and the bacteria extend into these spaces, spreading from thence inwards into the dentine.

The acid produced by the bacteria would very soon have the effect of inhibiting their growth, were it not that it is soon neutralized by the lime salts of the enamel.

In the same way any small quantities of acid frequently applied to the teeth will produce surface irregularities by solution of the interprismatic enamel substance, or of the axial prismatic portions, assisting in the adherence of organisms and forming foci for extension.

As an apparent contraversion of this principle, it may be argued

that those who habitually consume strongly acid fruit diet (such as the Sicilians) are particularly free from dental caries; in reality, however, this fact is not incompatible with the acid theory, for it is conceivable that the strong acid will inhibit the growth of the acid producing organisms, and, while it may dissolve the outer layers of enamel, secondary dentine will occlude the pulp chamber before that cavity is threatened.

Chief Predisposing Causes.

(*a*) *Structural Defects.*—Due to imperfect chemical constitution and congenital surface irregularities.

(*b*) *Crowding and Irregular Situation.*

(*c*) *Pregnancy.*

(*d*) *Inherited Tendency.*

(*e*) *Certain General Diseases* (such as gout and rheumatism).

(*f*) *Carbohydrate Diet.*

(*g*) *Acid tooth-powders.*

Classification of Etiological Theories.

(*a*) *Inflammatory.*—There are no blood-vessels in human dentine or enamel, and therefore inflammatory changes in them are impossible.

Gunshot wounds in tusks of elephants show no trace of inflammation in the damaged ivory, though the neighbouring pulp may be actually inflamed with abscess formation.

The incisors of rodents are liable to fracture, and several cases of re-union are recorded, but no sign of inflamed dentine has ever been detected in them.

The same may be said with respect to reported cases of fractured and re-united human teeth, which have shown no microscopical signs of inflammation.

Caries is able to continue in dead teeth as well as in living, and the process appears identical in human, or ivory, teeth, worn on plates as artificial substitutes.

This theory has been chiefly supported in recent years by *Abbott*, *Heitzmann*, and *Boedecker*, whose investigations appear to have received no support from other observers.

(*b*) *Chemical Theory.*—The stock arguments in favour of this theory are:—

1. Caries always starts from the surface.
2. The cavity gives an acid reaction.
3. Dilute acids dissolve enamel and dentine.
4. Saliva is generally slightly acid.
5. Artificial teeth, either natural or of ivory, are liable to decay and present the usual appearances of caries, such as the transparent zone, tobacco pipe stem, beading of the dentinal tubes, &c.

The argument against the theory is, that acids alone can never eproduce, artifically, all these appearances. Magitot has produced avities resembling those of caries in both enamel and dentine, by he action of sugar and acids; but a solution of the enamel alone ccurred when steps were taken to prevent fermentation by the ddition of antiseptics.

(*c*) *Electrical Theory.*—Bridgman advanced this theory, attribut-ng the phenomena of caries to the action of electric currents ecomposing the buccal juices. This is now only of historical nterest.

(*d*) *Parasitic Theory.*—Caries is certainly not a specific disease ue to one specific organism, though some writers have attributed e condition to *Protococcus dentalis*, which has been credited with e power of liquefying dentine and enamel. *Leber* and *Rottenstein* egard the earlier stages as chemical, and the later as due to the ction of *Leptothrix buccalis* penetrating the dentinal tubes.

Miller and *Underwood* consider that two factors are present—the ction of acids and germs.

Foodstuff Chemistry.—Fermentation is of such importance caries that fermentative changes may be briefly mentioned.

Foodstuffs consist of—

Proteids,
Carbohydrates, and
Fats.

The carbohydrate element is of the most importance in caries.

The carbohydrates can be divided into three main groups:—

1. Monosaccharides.
2. Disaccharides.
3. Polysaccharides.

1. *Monosaccharides*, $C_6H_{12}O_6$—

Comprising dextrose, levulose, and glucose.

They are—

(*a*) Found in nature in fruits, seeds, roots, and honey.

(*b*) Directly fermentable by yeast into alcohol and carbonic acid—

$$\underset{\text{Dextrose.}}{C_6H_{12}O_6} + \text{yeast} = \underset{\text{Alcohol.}}{2C_2H_5OH} + CO_2.$$

(*c*) Directly converted by bacteria into lactic acid—

$$\underset{\text{Dextrose.}}{C_6H_{12}O_6} = \underset{\text{Lactic acid.}}{2CH_3CHOHCOOH.}$$

2. *Disaccharides*, $C_{12}H_{22}O_{11}$—

Comprising cane sugar (sucrose), milk sugar (lactose), and malt sugar (maltose).

They are—

(*a*) Found in nature in sugar cane, milk, fruit, &c.
(*b*) Not directly fermentable by yeast.
(*c*) Inverted by a special ferment into dextrose and levulose, which are then fermentable by yeast.
(*d*) Inverted or fermented by certain mouth organisms, but is a slow process—

$$\underset{\text{Cane sugar.}}{C_{12}H_{22}O_{11}} + H_2O = \underset{\text{Dextrose.}}{C_6H_{12}O_6} + \underset{\text{Levulose.}}{C_6H_{12}O_6}.$$

$$\underset{\text{Cane sugar.}}{C_{12}H_{22}O_{11}} + H_2O = \underset{\text{Lactic acid.}}{4CH_3CHOHCOOH}.$$

3. *Polysaccharides*, $(C_6H_{10}O_5)_n$—

Comprising starches, cellulose, and the gums.

They are—

(*a*) Not directly fermentable by yeast.
(*b*) Fermentable by mouth and intestinal bacteria.
(*c*) Inverted to maltose by saliva ptyalin and by certain mouth bacteria—

$$\underset{\text{Starch.}}{2C_6H_{10}O_5} + H_2O = \underset{\text{Maltose.}}{C_{12}H_{22}O_{11}}.$$

With subsequent fermentation to lactic acid—

$$\underset{\text{Maltose.}}{C_{12}H_{22}O_{11}} + H_2O = \underset{\text{Lactic acid.}}{4CH_3CHOHCOOH}.$$

And conversion of some of the lactic acid to butyric acid by the action of anäerobic bacteria—

$$\underset{\text{Lactic acid.}}{2C_3H_6O_3} = \underset{\text{Butyric acid.}}{C_4H_8O_2} + 2CO_2 + 2H_2.$$

Of the non-carbohydrate foodstuffs we have :—

Proteids.—These undergo fermentation by the action of bacteria with the production of alkaloids and other nitrogenous substances.

Traces of propionic and other organic acids may be formed, but these have apparently but little importance in dental caries.

Fats.—The fermentation of fats in the mouth is exceedingly small, resulting in the formation of fatty acids, and probably playing little or no part in dental caries.

Notwithstanding the profound modification that environment and foodstuffs may have upon the flora of the mouth, the organisms present in caries are fairly constant, and are thus classified by Goadby :—

Bacteria of Dental Caries.

1. *Acid-forming Bacteria.*

Streptococcus brevis *Bacillus necrodentalis* *Staphylococcus albus*	Deep layers of carious dentine.
Streptococcus brevis *Sarcina lutea* *Sarcina aurantiaca* *Sarcina alba* (Eisenberg) *Staphylococcus albus* *Staphylococcus aureus*	Superficial layers of carious dentine.

2. *Bacteria liquefying decalcified Dentine.*

None isolated so far. Deep layers of carious dentine.

Bacillus mesentericus ruber *B. mesentericus vulgatus* *B. mesentericus fuscus* *B. furvus* *B. gingivæ pyogenes* *B. liquifaciens fluorescens motilis* *B. subtilis* *Proteus zenkeri* *Bacillus plexiformis*	Superficial layers of carious dentine.

From the foregoing review of these mouth conditions which are f such frequent occurrence in the tropics, it will be gathered that cid formation, and other action by the bacteria of the mouth, is esponsible for much of the mischief.

Especial care should, therefore, be taken by all dwellers in the ropics to see that the teeth are efficiently cleaned, and this by neans of some substances which are both antiseptic and, at the same me, alkaline in reaction.

The following prescriptions, recommended by *Goadby*, will be ound most excellent :—

℞.—Pulv. cret. precip., ...	ʒij.
Pulv. sap. dur., ...	ʒiv.
Pulv. iridis., ...	ʒiij.
Sod. carb., ...	ʒi.
Lysol, ...	♏x.
Extr. heliotrop. alb., ...	ʒss.

M.

Sig.—The tooth powder.

℞.—Lysol, ...	♏v.
Ess. piment., ...	♏x.
Sp. vini rect., ...	ʒij.
Aq. rosæ ad	ʒi.

M.

Sig.—The tooth wash.

CHAPTER V.

PREGNANCY AND INFANT-FEEDING IN THE TROPICS.

PREGNANCY.

So many wives leave the old country to follow the fortunes of their husbands in distant countries and colonies, and our tropical Empire contains so many millions of female, native fellow-subjects, that a chapter on the tropical aspects of child-bearing, although an innovation in a work of this sort, should nevertheless be acceptable to those whose public or private medical work will call, or has called, them far afield.

It may often fall to the lot of such practitioner to find that his work lies far from the beaten track, and that perhaps his nearest confrère is many miles distant.

Sir W. Sinclair has well said that "a young practitioner has learnt a lot of surgery that he will never practise, and will practise a lot of midwifery that he has never learnt." This aphorism is one which should form food for thought to the non-stay-at-home student, to whom a thorough working knowledge of routine midwifery and its eventualities, such as placenta prævia, transverse presentations, eclampsia, &c., may mean his success as a private practitioner, or perhaps may obviate many bitter reflections at the grave-side of his dearest friend's wife.

It is not, however, intended herein to discuss the normal phenomena of the pregnant state nor the mechanism of labour, but rather to briefly review certain influencing factors which justify the presence of these pages in a Manual of Tropical Medicine.

1. *Effects of environment and race.*
2. *Effects of heat.*
3. *Effects of tropical diseases.*

Having touched on these, the last part of the chapter will be devoted to *Infant-feeding.*

1. Effects of Environment and Race.—The European woman who forsakes home and all that gives that name its attractiveness to follow a husband to the outposts of the Empire finds herself launched into a new world absolutely beyond her ken. She is perhaps young and inexperienced. She is cast amongst strangers thousands of miles from all that is dear to her, perchance even beyond the confines of civilisation.

Apart from the differences of temperature almost every surrounding of life is new. The usually carpetless and scantily furnished

houses with their many doors and windows, generally open all the year round; the gloom of the jungle hinting of unknown mystery, or the dark-skinned inscrutable faces in their vivid setting; the silent, stealthy, impassive native servants; unknown fruits and novel cooking; the unaccustomed requirements of clothing—these and many other details must in themselves have a tremendous influence on her mental organisation, and when to this feeling of nostalgia is added the natural apprehension felt by most women who are about to reproduce their species, the reaction may be unfavourable, and may lead to a dangerous morbidness of mind.

Her native sister, on the other hand—born and bred on the spot—is free from the evil influences of this "mental environment." She has her friends around her. She is calm, phlegmatic, and unapprehensive, for which she has to thank that traditional fatalism with which the receding centuries have dowered her.

But her environment, if purely physical, is yet more deadly—an environment of ignorance and filth. She is entirely in the hands of the village midwife, or "handy woman," a prejudiced, ignorant, and dirty person. The yearly loss of infant life throughout the Empire must be appalling, and the day will surely soon come when legislation will be enacted to deal with this matter.

In all Colonies there is increasing concern for the welfare of our native fellow-subjects; efforts are being made to stamp out malaria and yellow fever. Sleeping sickness and beri-beri are claiming much attention, yet nothing is being done by Governments to stop the ravages of puerperal fever, trismus neonatorun, entero-colitis, &c., the causes and prevention of which are within our ken.

The dissemination of the knowledge of such preventive measures amongst natives, by means of trained native midwives, should not be an insuperable task; and such work could be supervised by the Colonial Authorities.

The mortality and morbidity from puerperal sepsis and the infantile death rate amongst native communities are enormous, and any money spent on education on these lines would result in a large economic gain to the community. Medical advisers to Colonial Administrations would therefore do well not to overlook the needs of those who play an unseen, but active part in the maintenance of our Empire.

With regard to the actual labour, it has often been stated that the child of civilisation has a bigger brain and a bigger head than the native child, and hence a more difficult labour for the white woman. This is not necessarily true. As a matter of fact the average weight of a native child is very nearly 1 lb. less than that of the European, but then the native pelvis is proportionally smaller than the European.

If, on the whole, there is less difficulty in native labour, the reason rather lies in the poor feeding on the part of the mother, and consequent malnutrition of the infant.

That the native woman suffers less during confinement, and has

fewer pathological complications during the puerperium than has the white woman, is not in accordance with the majority of tropical experience.

Finally, to summarise the foregoing remarks, we see that, for the native woman, under her native skies, want and poverty may play havoc with the nutrition of the child that is to be, but there is no adverse mental environment during the pregnant state; and that there is too often a physical environment of ignorance and sepsis during labour which demands the attention of public opinion.

On the other hand, for the European woman, more highly strung than her native sister, and situated under alien stars, the nine months is a period of mental strain which needs all the watchfulness and loving care of husband and familiar friends.

If situated far from the beaten track, with no neighbours of her own race and sex, and with a husband perhaps absent for most of each day, it is highly advisable at such a time that she should have the companionship of a sister or relation.

II.—Effects of Heat.—The European, coming from a temperate climate with a considerable annual variation and a low mean temperature, proceeding to the tropics where he finds a minimum variation and a mean temperature of perhaps 20° F. higher, is placed at a great disadvantage as compared to the native or acclimatised resident.

An immense strain is immediately put on the thermotaxic mechanism.

That section which regulates the loss of heat is continuously and energetically excited; while the heat producing section is more or less dormant and its considerable resources are a potential source of danger.

The frequent pyrexia which results from the condition of irritable weakness has been more or less fully dealt with in Chapter xviii. when discussing the subject of diathermasia.

But other phenomena result besides the liability to pyrexia.

There is a continuous expenditure of nerve energy in part by stimulating the processes for loss of heat, such as diaphoresis, &c., and in part by the constant inhibition of normal heat-production.

The result of this expenditure of nerve energy is to lower the nervous tension—a condition usually accompanied by lassitude and depression of spirits. To the pregnant female this is exceptionally unfortunate, for, even if not enceinte, her nervous system is much less able to ignore an unfavourable environment than is that of the male.

In women the nerve tension is normally lower than in men, and, consequently, any waste of nerve energy, though absolutely smaller in the less active female, is relatively much greater as she has the smaller initial capital.

The most obvious of the many effects of a hot climate during pregnancy are:—

(*a*) *Lassitude and Depression of Spirits.*—This, as we have seen

above, is due to the lowered nervous tension consequent on the strain placed on the thermotaxic mechanism by the unusual thermal environment.

(*b*) *Phosphaturia.*—*Fowlie* has found the condition in every pregnant woman under his care in Singapore during the last thirteen years.

This presence of phosphates in the urine of pregnant women is so rare at home that we can almost certainly look upon it as an effect of heat.

The lowered nervous tension, with an enfeebled system, leads to a perverted metabolism with an excessive excretion of phosphates.

(*c*) *Frequent occurrence of Pyrexia after Child-birth.*—The expenditure of nerve energy by heat production, &c., during labour is immense. At least four-fifths of the total energy expended during the long continued muscular effort of parturition appears as heat, and it is easy to conceive that the thermotaxic mechanism may often be unable to meet the emergency and pyrexia will result.

There are other consequences perhaps remotely connected with atmospheric heat and physical debility.

One of such is atonicity of the uterus, exhibited chiefly after labour in the form of *retarded involution.*

The *treatment* of all these effects is *absolute rest.* Herein we may learn much wisdom from the Oriental. His lethargy and love of repose, attributed by the lecturer to original sin, is in reality the lesson taught by the centuries that muscular exertion entails the necessity of getting rid of a superfluous production of heat.

Cases of phosphaturia are best treated by acid. phosph. dil.

℞.—Ac. phosph. dil.,	. . .	♏xx.
Sp. chlorof.,	. . . .	♏x.
Aq. cinnam, ad	. . . .	℥i.
M.—F. Mist. t. d. s.		

III. Effects of Tropical Diseases.—Tropical diseases incurred during the course of pregnancy may, or may not, have an effect on the mother.

The following table will give at a glance the conditions usually found to occur in practice:—

Cholera,
Plague,
Yellow Fever, if contracted during pregnancy, almost invariably lead to abortion: and, unless the attack is of the very mildest description, the maternal prognosis is almost hopeless.

Malaria.—If this disease is contracted during or after the sixth month of pregnancy, miscarriage is probable if the case be one of the malignant variety. The parasite may be transmitted to the infant by the maternal blood.

Leprosy.—Sterility and impotence, as a rule, are items in the course of the disease. If conception occur the child generally will abort at about the third or fourth month.

Beri-beri.—Abortions are infrequent, but pregnancy and parturition may predispose to the disease.

Amenorrhœa may result from many tropical disorders.

The Rearing of Infants.

Feeding.—During early childhood the European is not at the same disadvantage with regard to the heat as are the parents.

The thermotaxic mechanism is one of the latest of the automatic nervous mechanisms to be evolved. Consequently European and native children are much on a par for the first few years of their life, and it is not until the child's progressive development approximates it to the adult type that it becomes at a disadvantage as compared with the native child. When this happens a portion of the nerve energy which ought to be devoted to evolution and growth is wasted at a time when there is a minimum of reserve to fall back on.

This fact will explain the loss of health of most European children in most parts of the tropics after their fourth or fifth year.

But, while no worse off than the small dusky cousin in this respect, there is another problem to be faced, and that is the problem of infant-feeding.

A difficult problem anywhere, it is trebly difficult in the tropics. In so far as European babies are concerned the great difficulty lies in the fact that so few mothers are able to nurse their own children. The reason for this well-known circumstance is somewhat obscure; very possibly it may be a heat effect. Well-developed women, with large breasts and normal milk secretion, prove able to suckle their infant for a few days only. The baby should be given mother's milk as long as possible. As it diminishes it should be supplemented by a bottle, and, when the secretion has stopped, a bottle with prepared cow's milk alone should be recommended.

The usual lines of artificial feeding are:—

(*a*) Fresh animal milk adopted for infants' use—

Cow's.
Goat's.
Buffalo's.

(*b*) Tinned fresh milk.
(*c*) Tinned condensed milk.
(*d*) Patent foods.

As decomposition, due to the growth of bacterial life, is very rapid in the tropics, cleanliness of bottles, teats, or tubes used in infant-feeding is of the highest importance, and should not be entrusted to the native nurse.

With regard to the food to be given, class (*a*) above is the best in

nany circumstances. There are some points, however, which need :onsideration. The source of the cow's milk should be carefully ascertained. Cows in the tropics are often fed by their native owners on so much garbage that the milk is both poor and dele-.erious. The milk, moreover, is often watered with dirty water, or s carried in vessels not properly protected from the aerial impurities inseparable from a crowded tropical city.

If cow's milk is to be used, therefore, let it be from private cows, r else from a dairy farm which is above suspicion and which sends s milk in sterilised and sealed bottles.

As will be seen from the table of milk analyses, given in Appen-ix vi., goat's and buffalo's milk are richer than cow's milk, and hould be given only when cow's milk is not obtainable, and then nly after suitable preparation.

(*b*) *Tinned Fresh Milk.*—If properly and carefully put up, fresh ilk in tins is as useful as milk straight from the cow; indeed, nless the latter can absolutely be relied on, it is in most cases referable. The preservation of milk in these cases is secured either y sterilisation and hermetical sealing, or else by the addition of reservatives, such as boric acid, &c.

Amongst a certain number of excellent and reliable brands two nay be mentioned—

Gianelli Majno.
Dahl.

he former is the finest milk from the rich pastures of Lombardy; ie latter from various Norwegian dairy farms.

Fresh milk is usually diluted:—

Age.	Milk.	Water.
1 day to 1 month	1	2
1 month to 3 months	1	1
3 months to 4 months	1	$\frac{1}{2}$
4 months to 5 months	1	$\frac{1}{3}$
Above 5 months	1	Nil.

Add 50 grs. milk-sugar to each 5 oz. of diluted milk.
Add $\frac{1}{2}$ teaspoonful of cream to each oz.

Recent research in the clotting of milk has shown, however, that 'lk can be prepared for infant use without dilution.

Artus and Pages showed that milk which had been treated with ılates or fluorides did not curdle with rennet owing to the precipi-ion of lime salts effected thereby.

A. E. Wright, who made further investigations, showed that milk ts were formed in two different ways:—

1. *A firm clot, by action of rennet or human gastric juice.*
2. *A loose clot, by action of acids.*

He further demonstrated that, if the lime salts of the milk were precipitated by oxalates, the subsequent addition of rennet produced a clot of the second type.

As oxalates and fluorides are poisonous, Wright tried other precipitants for calcium salts, and found that sodium citrate answered all requirements.

It should be added :—

Sod. cit., gr. i. to each fl. ℥i. milk.

A flocculent digestible clot is thus formed in the child's stomach, and no dilution of the milk is necessary.

The antiscorbutic power of the citrate, its harmlessness, and its extreme solubility are additional arguments in its favour.

Note.—The sodium citrate can be conveniently obtained in the tabloid form.

With regard to the time and amount of feeding required, there is very little difference between the necessities of temperate and of tropical climates. Two night feedings should be given during the first month, and then only one until the sixth month, when they may be discontinued.

The infant's skin will act more freely in a hot atmosphere, to compensate for which a slightly increased amount of liquid may be given.

	Quantity for one Feeding.	Interval between Meals.
1st week,	1½ oz.	2 hours.
2nd ,,	2 ,,	2 ,,
3rd ,,	3 ,,	2 ,,
4th ,,	3½ ,,	2½ ,,
2nd month,	4 ,,	2½ ,,
3rd ,,	4½ ,,	2½ ,,
4th ,,	5 ,,	3 ,,
5th ,,	5½ ,,	3 ,,
6th ,,	6 ,,	3½ ,,

(*c*) *Tinned Condensed Milk.*—Unless no fresh milk is available, condensed milk should never be given. The large excess of cane sugar is quite unsuitable for the infantile digestion, and although a certain percentage of infants are strong enough to thrive on it, it is an experiment which were better avoided.

(*d*) *Patent Foods.*—There is, as a rule, no necessity for any patent food in the diet scheme of a healthy infant. Milk for the first six months of life is an ample dietary.

Occasionally it happens that, even after careful trial, milk does not agree. In this case it may be necessary to resort to an artificial substitute.

The physician is frequently asked to give his opinion on the relative merits of the various foods, and, as he is often unaware of what they are composed, the following table, taken from a lecture by Dr. Robert Hutchison, may prove of use. The dried human milk is the standard of composition to which artificial substitutes should conform :—

Food.	Water.	Proteid.	Fat.	Carbo-hydrate.	Salts.
1. Dried human milk, .	...	12·2	26·4	52·4	2·1
2. Allenbury, No. 1, .	5·7	9·7	20·0	60·85	3·75
3. Allenbury, No. 2, .	3·9	9·2	15·0	69·1	3·50
4. Mellin, . . .	6·3	7·9	trace	82·0	3·8
5. Savory & Moore, .	4·5	10·3	1·4	83·2	0·6
6. Benger, . . .	8·3	10·2	1·2	79·5	0·8
7. Allenbury, No. 3. .	6·5	9·2	1·0	82·8	0·5
8. Moseley's, . .	10·8	11·0	0·92	76·4	0·94
9. Ridge's, . . .	7·9	9·2	1·0	81·2	0·7
10. Neave's, . . .	6·5	10·5	1·0	80·4	1·6
11. Frame food, . .	5·0	13·4	1·2	79·4	1·0
12. Robinson's groats, .	10·4	11·3	1·6	75·0	1·7
13. Robinson's patent barley, . .	10·0	5·1	0·9	82·0	1·9

1. The standard.

2. A desiccated cow's milk, from which the excess of casein has een removed, and a certain proportion of soluble vegetable albu-nen, milk, sugar, and cream added. No starch is present. ℥ss. in iij. of water for a child aged three months.

3. As above, but contains malted flour in addition. No starch resent. ℥i in ℥vj. water for a child of six months.

4. A completely malted food. The carbohydrate is in a soluble orm. 5·0 grammes (half-tablespoonful) to ¼ pint of milk and pint of water—for child under three months. Deficient in fat.

5. Wheat flour with added malt. If prepared by directions most f the starch is converted into soluble dextrins. Deficient in fat. ne tablespoonful with two of milk ; mix ; and add ⅓ pint of oiling milk and water.

6. Wheat flour and pancreatic extract. Most of the starch is ade soluble during preparation. Proteid partly digested. One blespoonful mixed with four of cold milk. Add ½ pint boiling ilk and water. Keep in warm place for fifteen minutes, and then ing to the boil. Deficient in fat.

7. Wheat flour and malt. Most of the starch is altered during eparation. Deficient in fat. Intended for children of over six

months. One tablespoonful, one teaspoonful of sugar, three tablespoonfuls of cold water; mix, and add ½ pint of boiling milk and water.

8. Complete conversion of starch occurs during preparation. To be given with milk.

9. A baked flour with only 3 per cent. of soluble carbohydrates. Recommended to be made with milk *or* water. If with the latter it is *not* a sufficient food.

10. Much as above.

11. Baked flour with cane sugar and bran. Abundant nitrogenous matter and much unaltered starch.

12. Ground oats minus the husk. Rich in proteid and salts.

13. Ground pearl barley.

For infants under six months of age no unaltered starch is permissible; and under three months no malted food. The two Allenbury Foods (Nos. 1 and 2) are the only ones that meet requirements of proper standards, having no starch, and a proper amount of fat.

General Management.—The infants should be bathed regularly—twice daily.

The navel cord should be dressed daily with some unirritating antiseptic dressing until it has separated. Lack of this precaution has resulted in tetanus neonatorum and septic invasion; the utmost attention should therefore be given to it.

Clothing of the new born infant should be warm, but not of flannel, which is very irritating.

As the child grows up, a minimum of clothing is required in the tropics. The legs and feet should be bare, or open sandals worn.

Dusting powders for infants should be sparingly used. Fuller's Earth is the safest and best. The soap should be unirritating, and should be sparingly used.

One does not know how much prickly heat can be produced by too much soap, too much powder, and too much clothing.

During childhood let plenty of open air exercise be taken; even during the heat of the day a child will be none the worse for being out in the compound, if the head is properly protected by a light but efficient topi.

Above all let the children have plenty of sleep: ten to twelve hours per diem are none too much.

Empirically, a routine dose of grey powder once a month is admirable for all European children.

Finally, have children sent home at seven years of age.

CHAPTER VI.

CLASSIFICATION OF ANIMAL PARASITES.

(ZOÖ PARASITES.)

In this chapter it is not intended to give any detailed description of the morphology or life history of the immense number of zoo parasites — either temporary or permanent, either epizootic or entozootic—which show partiality for long-suffering human beings, already bowed down with "the weariness, the fever, and the fret."

Those of the greatest pathological importance will be found to be adequately treated elsewhere.

Of the **Protozoa**, Chapter xxxv. deals with the *Trypanosomes*; Chapter xxvi. with the *Leishman-Donovan bodies*; Chapter xxviii. with the *malarial parasite*.

In Chapter viii. will be found an account of the general morphology and type history of the **Cestodes, Nematodes**, and **Trematodes**; and the latter discussed even more fully in Chapters xiv. and xix. on *Bilharziosis* and *Distomiasis*; and *Filariæ* (nematodes) in Chapter xii., and the *Guinea-worm* in Chapter xx.

Of the **Arthropoda**, Chapter x. deals with the *Ixodidæ* (or ticks), and the *Aphaniptera* (or fleas).

The morphology, life history, and detailed classification of the *Culicidæ* forms the subject of Chapter ix.; while in Chapter xi the venomous bites of many noxious species are briefly reviewed. Mosquitoes, flies, and other transient biters can hardly come within the designation of *parasites* for the purposes of this list.

The following is merely an outlined list of the natural history divisions, classes, orders, genera, and species of the chief zoo parasites (after *Braun* and *Nicholson*), which may prove of use in looking up the biological status of the various species which are so commonly met with in the pathological work of the tropics:—

Sub-kingdom—PROTOZOA.

Division or Class.	Order.	Family.	Genus.	Species.
Rhizopoda.	**Amœbina.**		*Amœba.*	*Amœba coli.*—(*vide* Chap. xxi.) *Amœba urogenitalis.*—Found in vaginal cysts and urine on several occasions 0·05 mm. Very motile. *Paramœba hominis.*—Observed in Philippines. Flagellated at one stage of growth. Causes diarrhœa.
Flagellata.	**Polymastigina.**		*Trichomonas.*	*Trichomonas vaginalis.*—Acid vaginal mucus. Three united flagella. Length, 0·02 mm.
			Lamblia.	*Cercomonas intestinalis.*—Found in small intestine. Length, 0·02 mm. Shape, oval. Dumb-bell nucleus. Three pairs of flagella.
	Protomonadinæ.	**Trypanosomidæ.**	*Trypanosoma.*	Many species known. See Chap. xxxv. Have an undulating membrane and one flagellum.
			Trypanoplasma.	Have both an anterior and a posterior flagellum.

				Oval. 0·04 mm. long. Habitat, liver. Undergoes development much like the malarial parasite. Five reported cases in man. *Coccidium hominis.*—Habitat, intestinal epithelium. Three reported cases in man. *Coccidium bigeminum.*—Diameter, 0·015 mm. Habitat, intestinal villi. Oocyst divides in two, becomes encysted, and each forms four spores. One reported case in man.
	Hæmosporidia.	Hæmamœbida.	*Hæmamæba.*	*Hæmamæba vivax.*—Parasite of benign testian malaria. *Hæmamæba malariæ.*—Parasite of quartan fever. *Hæmamæba precox.*—Parasite of malignant malaria. See Chap. xxvii.
Infusoria.	Heterotricha.		*Balantidium.*	*Balantidium coli.*—Oval. 0·1 mm. long. Habitat, large intestine. 63 human cases recorded. *Balantidium minutum.*—Oval. 0·025 mm. long. Fissure-like peristome extends to centre of body. Habitat, small intestine. Found twice in man.

Sub-kingdom—**ANNULOSA.**

Division or Class.	Order.	Family.	Genus.	Species.	
Platyelmia.	**Cestoidea.**	**Bothriocephaloidea.**	*Dibothriocephalus.*	*D. latus.*	See Chap. viii.
				D. cordatus.	
			Diplogonoporus.	*D. grandis.*	
				D. mansoni.	
		Tæniidæ.	*Dipylidium.*	*D. caninum.*	
			Hymenolepis.	*H. nana.*	
				H. diminuta.	
				H. lanceolata.	
			Davainea.	*D. madagascariensis.*	
			Tænia.	*T. solium.*	
				T. marginata.	
				T. serrata.	
				T. crassicollis.	
				T. saginata.	
				T. africana.	
				T. confusa.	
				T. echinococcus.	
	Trematoda	**Paramphistomidæ.**		*Gastrodiscus hominis.*	See Chap. xix.
		Fasciolidæ.	*Fasciola.*	*F. hepatica.*	
			Fasciolopsis.	*F. buski.*	
			Paragonimus,	*P. westermani.*	

			Opisthorchis.	*O. felineus.* *O. sinensis.* *O. noverca.* — See Chap. xix.
			Cotylogonimus.	*C. heterophyes.* — See Chap. xix.
			Dicrocœlium.	*D. lanceatum.* — See Chap. xix.
		Schistosomidæ.	*Schistosomum.*	*S. japonicum.* — See Chap. xix. *S. hæmatobium.*—See Chap. xiv.
Nematelmia.	Acanthocephala.		*Echinorhynchus.*	*E. gigas.*—Gutless worm, with 6 rowhooked rostrum. Male, 12 cm.; female, 40 cm. Habitat, intestine. Common in pig. Rare in man. Cockchafer is intermediate host. *E. hominis.*—Rostrum has 12 rows of hooks. Only 5·5 mm. long. Habitat, intestine.
	Nematoda (see Chap. viii.)	Anguillulidæ.	*Rhabditis.*	*R. pellio.*—Male, 1 mm.; female, 1·1 mm. Larval stage in earthworms. Adult stage in decomposing soil. Facultative parasite of human vagina.
		Angiostomidæ.	*Strongyloides.*	*S. intestinalis* (or *Anguillula stercoralis*).—Has two generations:— (1) Parasitical— Habitat, intestine. Worms hermaphrodite. Eggs develop in intestine. Young passed out with fæces. Adults measure 2·2 mm.

Sub-kingdom—**ANNULOSA**—*Continued.*

Division or Class.	Order.	Family.	Genus.	Species.
Nematelmia (*continued*).	**Nematoda** (*continued*).	**Angiostromidæ** (*continued*).	*Strongyloides* (*continued*).	(2) Extra corporeal— Males, 0.7 mm. Tails coiled. Female, 1 mm. Eggs laid and hatch into rhabditic form; then moult and change into strongyloid form.
		Filariidæ.	*Filaria.*	*F. medinensis* or Guinea-worm.—See Chap. x.
				F. bancrofti. *F. loa.* *F. perstans.* *F. demarquayi.* *F. ossardi.* *F. magalhaesi.* *F. philippinensis.* } See Chap. xxii.
		Trichotrachelidæ.	*Trichocephalus.*	*T. dispar.*—Whip-worms. Male, 45 mm.; female, 50 mm. World-wide distribution. Habitat, cæcum. Eggs developed in water or moist soil.

			Trichinella.	*T. spiralis.*—Black and brown rats normal hosts. Pigs and rabbits easily infected. Encysted trichinellæ ingested; capsules dissolved; become adult in duodenum; copulate; males die; females grow to 3 mm.; bore into villi; 1,500 young discharged into lymph spaces and carried through body; young then leave capillaries and invade muscle. This destination reached only 10 days after ingestion of cystic parasite by host. Females die in 7 weeks. Embryos remain alive in the cysts for 25 to 31 years.
		Strongylidæ.	*Eustrongylus.*	*E. gigas.* — Blood-red worm. Males, 40 cm.; females, 100 cm. Habitat, pelvis of kidney. Hosts, dog, horse, &c. 12 human cases reported
			Strongylus.	*S. apri.*—Males, 25 mm.; females, 50 mm. Tail of female re-curved. Habitat, bronchial tubes of pigs. Eggs elliptical. Infection direct. One or two human infections reported

Sub-kingdom—**ANNULOSA**—*Continued.*

Division or Class.	Order.	Family.	Genus.	Species.
Nematelmia (*continued*).	**Nematoda** (*continued*).	**Strongylidæ** (*continued*).	*Strongylus* (*continued*).	*S. subtilis.*—Male, 5 mm.; female, 7 mm. Eggs oval. Habitat, intestine of camel. Found in man in Egypt.
			Ankylostoma.	*A. duodenale.*—See Chap. xii.
		Ascaridæ.	*Ascaris.*	*A. lumbricoides.*—Round worms. Male, 20 cm.; female, 30 cm. World - wide distribution. Habitat, small intestine. Ova develop in water and moist earth, Infection direct.
			Oxyuris.	*O. vermicularis.*—Thread-worms. Male, 4 mm.; female, 10 mm. World - wide distribution. Habitat, large intestine. Infection direct.
Arthropoda.		**Ixodidæ.**	*Rhipicephalus.*	*R. annulatus.* *R. caudatus.* *R. evertsi.* *R. decoloratus.* *R. australis.* *R. sanguineus.* *R. pulchellus.* See Chap. x.

			Hæmaphysalis.	*H. leachi.*	See Chap. x.
			Dermacentor.	*D. electus.*	
			Ixodes.	*I. ricinus.*	
				I. hexagonus.	
			Amblyomma.	*A. variegatum.*	
			Hyalomma.	*H. ægyptium.*	
		Argasidæ.	*Argas.*	*A. reflexus.*	
				A. persicus.	
			Ornithodorus.	*O. moubata.*	
		Tyroglyphidæ.	*Glyciphagus.*	*Glyciphagus buski.*—Mite causing craw-craw. See Chap. xxxi.	
		Sarcoptidæ.	*Sarcoptes.*	*Sarcoptes scabei* or *Acarus scabei.*—See Chap. xxxi.	
		Demodicidæ.	*Demodex.*	*D. folliculorum.*—See Chap. xxxi.	
Insecta.	Anoplura.	Pediculidæ.	*Pediculus.*	*P. capitis.*—Head louse.	
				P. vestimenti.—Body louse.	
				P. pubis.—Crab louse.	
	Rhynchota.	Geocorisæ.	*Cimex.*	*C. lectularius.*—The bed bug.	
			Conorhinus.	*C. sanguisuga.*—Texas bed bug.	
	Aphaniptera.	Pulicidæ.	*Pulex.*	*P. irritans.*	See Chap. x.
				P. cheopis.	
				P. serraticeps.	
				P. fasciatus.	
				P. philippinensis.	
				P. anomalus.	
			Hystrichopsylla.	*H. obtusiceps.*	
			Typhlopsylla.	*T. musculi.*	
				T. assimilis.	
			Sarcopsylla.	*S. penetrans.*	
				S. gallinacea.	

CHAPTER VII.

CLASSIFICATION OF VEGETABLE PARASITES.

THE vegetable world is generally divided into two great kingdoms—the Cryptogams and the Phanerogams.

The outline classification of these is as follows :—

Cryptogamia :—

GROUP I.—Thallophyta.

Class I. Fungi (devoid of Chlorophyll).
,, II. Algæ (containing Chlorophyll).

GROUP II.—Bryophyta.

Class I. Hepaticæ (Liverworts).
,, II. Musci (Mosses).

GROUP III.—Pteridophyta.

Class I. Filicinæ (Ferns).
,, II. Equisetinæ (Horsetails).
,, III. Lycopodinæ (Lycopodiaceæ).

Phanerogamia :—

GROUP I.—Gymnospermæ (mostly evergreen trees and shrubs).

Class. Gymnospermæ.

GROUP II.—Angiospermæ.

Class I. Monocotyledones.
,, II Dicotyledones.

The above classes are further subdivided into innumerable cohorts, orders, &c.

When reviewing the animal parasites of man, it will have been noticed that very many natural history orders contributed to the list, since man is himself an animal.

With vegetable parasites it is very different; and the lowest class (Fungi) of the lowest vegetable group (Thallophyta) alone contributes to the rôle of human parasitism. The following is the best classification of this class—Fungi :—

Group.—THALLOPHYTA.

Class.—Fungi.

ORDER I. *Schizomycetes* (including all the bacteria). These are unicellular organisms. Reproduction usually by fission.

FAMILY 1. *Coccaceæ* (spherical forms).

Genus 1. Micrococci (division irregular).
Genus 2. Streptococci (division in one plane).

FAMILY 2. *Bacteriaceæ* (rod forms).

Genus 1. Bacteria (no spore formation).
Genus 2. Bacillus (spore formation).
Genus 3. Spirillum (spiral form. No spores).
Genus 4. Vibrio (spiral form. Spores).

FAMILY 3. *Leptotricheæ* (unbranching threads).

FAMILY 4. *Cladotricheæ* (Pseudo-branching threads).

FAMILY 5. *Streptotricheæ* (Dichotomous branching threads).

ORDER II. *Blastomycetes* (including all the yeasts). These are unicellular organisms. Reproduction by budding.

ORDER III. *Hyphomycetes* (including all the moulds). These are multicellular organisms. Reproduction by spores, and is both asexual and sexual (rudimentary).

Chief Pathogenic Species.

I.—OF THE SCHIZOMYCETES.

(*a*) Genus *Micrococcus.*

M. pyogenes aureus.

Is an aërobe—and a facultative anaërobe. Non-motile. No spores. Liquefies gelatin. Stains by Gram. Commonest organism of suppuration.

M. pyogenes albus.

Morphology as above. Less common than *aureus.* Found in wounds and panophthalmitis.

M. gonorrhææ.

Aërobic. Slightly motile. No spores. Only grows in native proteid. Not stained by Gram. Peculiar to man. Occurs in pairs in the pus cells.

M. melitensis.

Aërobic. Slightly motile. No spores. No liquefaction of gelatin. Decolourised by Gram. Specific cause of Malta fever (see Chapter xxix.)

M. zymogenes.

Aërobic. Non-motile. No spores. Slow liquefaction of gelatin. Stained by Gram. Curdles milk in twenty-four hours and later liquefies the curd, forming a turbid reddish liquid. Has been found in acute endocarditis.

M. tetragenus.

Aërobic. Non-motile. No spores. Does not liquefy gelatin. Stained by Gram. Met with in phthisical cavities, and in some abscesses. General infection has been recorded.

M. pneumoniæ (Fränkel).

Oval coccus. Aërobe and facultative anaërobe. Does not liquefy gelatin. Stained by Gram. Pathogenic for mice, rabbits, and guinea-pigs. Is specific agent of acute pneumonia. Occurs in pairs. In cultures usually as a short streptococcus. Is a pyogenic organism. Frequently found in meningitis and otitis media.

(*b*) Genus *Streptococcus.*

S. pyogenes aureus.

Aërobic. Non-motile. No spores. Does not liquefy gelatin. Stained by Gram. Is the only organism that does not reduce weak methylene blue solution. Found in *abscesses* and *lymphangitis.* Is specific cause of *pyæmia* and *puerperal fever.* Causes one-third of the cases of acute infective *endocarditis.* Causes *septic pneumonia* after mouth operations. Causes *erysipelas*, being identical with *S. erysipelatis.*

S. scarlatinæ.

Morphologically not unlike *S. pyogenes*, but produces much acid and marked curdling of milk. Is special agent of *scarlatina.* Is pathogenic to mice.

(*c*) Genus *Bacterium* (non-spore forming).

B. friedländeri.

Short encapsuled rod. Aërobe. Facultative anaërobe. Non-motile. No spores. Decolourised by Gram. Does not liquefy gelatin. Pathogenic to mice and guinea-pigs. Rabbits immune. Occasionally found in pneumonias.

B. aerogenes capsulatus.

Large encapsuled bacterium. Non-motile. Stained by Gram. Anaërobe. Facultative aërobe. Liquefies gelatin. Gas forming. Widely distributed in soil,

dust, intestines, &c. Found in emphysematous gangrene, perforative peritonitis, &c.

B. mallei.

Small bacterium. Non-motile. Aërobe. Facultative anaërobe. Does not liquefy gelatin. Decolourised by Gram. Causes *glanders* in horses. Man affected on hand, arm, or nasal mucous membrane: if acute, there is fever, delirium, and death; if chronic, indolent ulcers.

B. diphtheriæ (Klebs-Löffler).

Delicate bacterium, with rounded ends, generally clubbed at one end. Non-motile. Stained by Gram. Does not liquefy gelatin. Aërobe and facultative anaërobe. Found in diphtheritic membranes.

B. typhosus (Eberth).

Short rods with rounded ends. Flagellated and actively motile, flagella numbering 8 to 12. Decolourised by Gram. Aërobe and facultative anaërobe. Does not liquefy gelatin. Forms acid in bile salt broth. Found in cases of typhoid fever in Peyer's patches, mesenteric glands, spleen, and urine.

B. coli commune.

Short rod with rounded ends. Has 3 or 4 flagella. Motile. Decolourised by Gram. Aërobe and facultative anaërobe. Does not liquefy gelatin. Forms acid and gas in bile salt broth. Most widely distributed organism in nature. Constant inhabitant of intestinal tract. Has been found in ischio-rectal abscess. Is common cause of cystitis.

B. enteritidis (Gartner).

Morphologically resembles *B. typhosus*. Forms neither acid nor gas in bile salt broth. Sometimes causes meat poisoning. Motile. Does not liquefy gelatin. Decolourised by Gram.

B. influenzæ (Pfeiffer).

Very minute rod. Aërobic. Non-motile. No growth on gelatin. Decolourised by Gram. Found in sputum and nasal secretions at height of disease Pathogenic only to monkeys and rabbits.

B. pestis (Kitasato).

Short ovoid bacterium. Marked polar staining. Two terminal flagella. Non-motile. Decolourised by Gram. Does not liquefy gelatin. (See Chapter xxx.)

(*d*) Genus *Bacillus* (forming spores).

B. anthracis (Pollender).

Large rod-shaped bacillus. Aërobe and facultative anaërobe. Non-motile. Stained by Gram. Liquefies gelatin. Spores highly resistant. Pathogenic to man, cattle, rabbits, mice, &c. Known as "splenic fever" in cattle. Occurs among wool sorters, either as "*malignant pustule*," or as a general infection.

B. tuberculosis (Koch).

Slender rod-shaped bacillus with rounded ends. Aërobe and facultative anaërobe. Non-motile. Does not liquefy gelatin. Stained by Gram. Spore formation highly probable. Grows well on nutrient agar and glycerin. Most domestic animals non-immune.

B. lepræ (Hansen).

Resembles *B. tuberculosis*, but more slender. Probably forms spores. Non-motile. No growth on gelatin. Stained by Gram.

B. tetani (Nicolaier).

Straight slender rod with rounded ends Spores give "drumstick" appearance. Anaërobic. Motile. Liquefies gelatin. Stained by Gram. Found in some soils. Exists only in wound, all symptoms being due to toxins produced.

B. œdematis maligni (Pasteur).

Long, slender, flagellated rod. Motile. Liquefies gelatin. Decolourised by Gram. Anaërobic. Develops foul smelling gas. Occurs in septic wounds and fractures. Causes putrefactive, emphysematous œdema.

(*e*) Genus *Spirillum* (no spores).

S. choleræ (Koch).

The "Comma bacillus." Curved rod. Single terminal flagellum. Aërobe and facultative anaërobe. Motile. Liquefies gelatin slowly. Decolourised by Gram. Produces acid in bile salt broth. (See Chapter xvi.)

S. finkler-priori.

Morphology as above. No indol with H_2SO_4 alone. Pathogenic to guinea-pigs. Liquefies gelatin very fast. No acid in bile salt broth.

S. metschnikovi.

Morphology much as above. Acid in bile salt broth.

Note.—Certain blood and tissue *Spirilla*, such as *S. obermeieri*; *S. duttoni*; *S. pallida*; *S. frambæsiæ*, are frequently placed in this group, but recent research rather lends colour to the theory of an animal origin akin to the protozoa (trypanosomes, &c.).

Family Leptotrichæ.

These occur as mouth organisms, none of them being pathogenic. The chief are :—

Leptothrix innominata.

L. buccalis maxima.—They have only been met with in dental, bacteriological research.

L. tonsillaris has been found in white pharyngeal patches in man.

Family Cladotricheæ.

This family consists of one genus only, but there are at least two species of pathological import to man.

Cladothrix Actinomyces.

Has long been known in cattle. Forms nodules not unlike those of tuberculosis. Found in the jaw, tongue, liver, lungs, &c. Liquefies gelatin. Is stained by Gram. Benefitted by pot. iod. Easily reproduced in lower animals.

C. mycetomæ—

Is a localised disease of the extremities in human beings. Commonly known as "Madura-foot." The foot or leg is enlarged, and numerous sinuses. The bones are carious. The soft structures are tough and hypertrophied from chronic inflammation. Cavities are present, containing black or yellow granules. Does not liquefy gelatin. Is decolourised by Gram. Pot. iod. has no effect. Lower animals are immune.

III. Of the Blastomycetes.

These are divided into two families :—

1. *Saccharomycetes* (spore formation).
2. *Torulæ* (no spores).

The former alone has any pathological significance, or is parasitic in man.

Of these the following are of interest :—

Saccharomyces albicans (Thrush disease).

Associated with white patches in tongue and mouth of infants. These patches consist of a mixture of yeasts,

moulds and bacteria. Does not liquefy gelatin. In saccharine media the growth is only cellular. If non-saccharine and an old culture, mycelial filaments occur.

S. litogenes (Sanfelice).

Has been found associated with malignant growths in men and animals. If inoculated in guinea-pigs death occurs with formation of a somewhat calcified primary tumour, and secondary embolic growths in lungs, spleen, and mesenteric glands. Does not liquefy gelatin. Aërobic. No growth on serum. Is probably the cause of the eighteen reported instances of a blastomycetic dermatitis.

III. Of the Hyphomycets.

Amongst the well-known genera of this order are :—

1. *Penicillium.*
2. *Mucor.*
3. *Oidium.*
4. *Aspergillus.*
5. *Microsporon.*
6. *Trichophyton.*
7. *Achorion.*

Of these, only the latter four are of pathological import to man.

Aspergillus fumigatis.

Has been found in man, especially amongst bird fanciers. Occurs as a pneumomycosis and also in otitis media. Grows well on laboratory media.

Microsporon audouïni.

Causes most of the scalp ringworm in England. Rarer towards East. Occurs as a white sheath round the stumps of broken hairs. The mycelial filaments occupy the interior of the hair.

Microsporon mansoni (Castellani).

The cause of Pityriasis nigra. Circular desquamating black patches. Little or no itching. Never on face. Most frequently neck. Mycelial threads short and not branching. Spores globular, large and arranged in clusters.

Microsporon furfur.

Cause of Pityriasis versicolor. Has very much the character of *Oidium lactis.* A mycelium with true hyphæ, ending in conidia. Does not liquefy gelatin.

Trichophyton endothrix.

Causes a certain number of the European and Eastern skin and scalp lesions. Hairs swollen and dark; no parasitic sheath.

Trichophyton ectothrix.

Causes the common body ringworm of the tropics (Tinea circinata) and possibly also dhobie itch.

Trichophyton mansoni.

The cause of Tinea imbricata.

Trichophyton pictor (Blanchard).

The cause of Pinta.

Achorion schönleini.

The cause of Favus.

Note.—For more detailed descriptions of the above *vide* Chapter xxxi. on the skin disease of the tropics.

CHAPTER VIII.

NOTES ON CESTODES, TREMATODES, AND NEMATODES.

A LARGE number of the flat- and round-worms of the sub-kingdom Annulosa are parasites of man.

In this chapter it is proposed to give a brief *résumé* of the structure, development, and biology of the Cestodes, Trematodes, and Nematodes; followed by a list of the chief species which are parasitic to man.

In Chapter xix. the chief species of human trematodes will be found to be more or less fully dealt with; and in Chapter xxii. the filarial family of the nematodes will be considered in detail.

CESTODES (Tape-worms).	TREMATODES (Flukes).	NEMATODES (Round-worms).
1. Skin cellular, . .	Skin chitinous, .	Skin chitinous.
2. No alimentary canal, . .	Mouth and blind alimentary canal,	Mouth, canal, and anus.
3. Hermaphrodite, .	Hermaphrodite (except *Bilharzia*),	Unisexual.

CESTODES (Rudolphi), 1809.—Human tape-worms have been known from ancient times. The evacuated proglottides—known as *Vermes cucurbitani*—were thought to unite in the body to form a tape-worm.

In 1683 the tape-worm's head was discovered by *Tyson* in a specimen from a dog; and, as a consequence of this discovery, the tape-worm became considered as a single animal.

The researches of *Steenstrup* in 1841 led to a further stage of advance by propounding the view of "alternation of generations."

More work on the subject was done by *v. Siebold* and *Leuckart*; and, until recently, these views were generally adopted.

Anatomy.—The whole tape-worm (or strobila) is covered with a cellular elastic *cuticle*, covering the suckers, and being reflected inwards at the genital orifices.

Immediately below this is the *parenchymatous basal membrane* nd dermo-muscular elements, which, together, form the chief ssue of the body.

The *cuticular glands* are scarce.

The *scolex* is the anterior segment from which are developed all ie proglottides, and which serves to fasten the tape-worm to the irface of the intestinal wall. At the anterior end are situated arious organs which carry out this function:—

1. *The Suckers*—usually four round acetabula, placed crosswise . the periphery of the thickened end of the scolex. They are arts of the musculature formed by a powerful development of the orso-ventral muscles.

In the Bothriocephaloidea, the place of the four round suckers taken by two groove-like structures, which answer the same urpose.

2. *The Hooklets.*—These are of varying number and situation. hey may be on the suctorial organs or above them. In many ecies they appear in a circular arrangement around a single otractile organ—the *rostellum*. Sometimes the latter is rudimen-ry, and is replaced by a terminal sucker.

The thickened part of the scolex which bears the suckers is ually called the *head*.

The flat part which connects the head with the proglottides is lled the *neck*.

The *proglottides*, or body segments, are joined to the scolex in a ngitudinal row, the distal segment being the oldest. The number ries, according to the species, from one to several thousands. As rule, the youngest segments are transversely oblong, the central es more inclined to be square, and the distal ones longitudinally long.

The anterior border of each proglottis fits into a groove in the sterior border of the preceding one.

The *nervous system* begins in the scolex as a kind of central rvous system consisting of medullary fasciculi united by commis-res. The system runs through the neck and the entire series of oglottides. The peripheral nerves arise from these fasciculi, of ich some lead to the muscles, and others (passing between the cuticular cells) terminate at the cuticle in a small plate, supposed be a sense-organ.

The *excretory apparatus* consists of terminal cells distributed efly in the cortical layers of the parenchyma. These are con-cted by either straight or tortuous capillaries, with four muscular l epithelial lined canals, which run the whole length of the worm, o on each lateral border.

In the scolex these vessels are united by a loop.

In the terminal segment they open into a short fusiform bladder, ich discharges in the middle of the posterior edge. When this ginal terminal proglottis has been shed, the longitudinal branches charge separately.

The excretory product consists of a clear fluid containing a substance similar to a solution of guanine and xanthine. Regurgitation is prevented by valves.

The Generative Apparatus.—One genus (*Diæcocestus*) is said to be sexually differentiated. All the other cestodes are hermaphroditic.

The sexual organs are situated in the medullary layer of each proglottis. They are not found in the younger proglottides near the scolex, but develop gradually in the more mature segments—the male organs forming earlier than the female.

The *male* element consists of a large number of testes, each of which is connected with a centrally placed vas deferens by vasa efferentia. The orifice of this vas deferens lies near the vaginal opening within a genital atrium, of which the raised border projects either at the edge of the proglottis or ventrally, to form the genital pore; this orifice of the vas is termed the cirrhus or penis.

The *female* element, commencing from the vaginal opening within the genital atrium, proceeds inwardly, assuming a spindle-shaped form (receptaculm seminis), and being continued as a spermatic duct to join the oviduct, or common excretory duct of the ovaries.

The ovaries are two tubular glands lying in the posterior half of the proglottis.

After the junction of the oviduct with the spermatic duct, the canal continues as a duct for fertilisation, and after a short course receives the ducts of the vitellogene and shell glands.

The fertilised ova, with masses of vitellus, receive the shell material at the entry of the shell gland duct, and pass on to the uterus as completed eggs.

Life History.—The ovum, as seen in a ripe proglottis, or free in fæces or water, consists of a hard envelope containing an embryo, sometimes termed a "hexacanth embryo," since six rudimentary hooklets are found at one end. It is also known as an "oncosphere."

This ovum is ingested by an *intermediate host*, the chitinous covering is dissolved and the embryo set free.

It bores its way through the intestinal wall with its hooklets and attains its destination in some muscle or organ.

Here one of four phenomena may occur:—

1. It may produce a cyst with germinal epithelium, which in turn produces other cysts in which scolices develop. This is called an *Hydatid cyst;* or

2. The embryo may produce a single cyst, eventually forming a scolex. This is a *Cysticercus.*

3. Only a rudimentary cyst may be formed. This is a *Cystoidcus.*

4. The embryo may not be surrounded by any cyst (as in *Bothriocephalidæ*), but merely elongate and form a scolex.

This development within an intermediate host does not, however, complete the life cycle of the cestode.

No further development will take place in these situations, and,

lthough they may remain alive for some months, they will ultimately alcify.

If, however, they are ingested (a second time in their history) by ome *terminal host*, then further development takes place. The berated scolex reaches the intestine of its new host, attaches itself) the mucous membrane, and immediately commences to form an dult worm by growth of proglottides.

In this way the cysticerci of mice and rats (*C. fasciolaris*) reach e intestine of cats; those of the hare and rabbit (*C. pisiformis*) ach the gut of dogs; those of the pig (*C. cellulosæ*) reach the uman intestine, &c.

Grassi and *Rovelli* have, however, shown that the intermediate ost is not always necessary, for the *Tænia murina* of rodents, when the cysticercus stage, is embedded in the intestinal wall; and reaking into the lumen, the scolex develops there into the adult worm.

The time required by the scolex to complete its chain of pro- ottides does not depend on the number it has to produce. *T. hinococcus*, with its three or four segments, takes the same eleven twelve weeks to grow as does *T. solium*, with its multitude of oglottides.

The duration of an adult tape-worm's life is variable. In most ecies it may be placed at about one year; in others it averages ly a few days; or, again, some will live for several years.

Natural death occurs by loss of hooklets, atrophy of suckers, &c.

Classification (after Braun).

I. Bothriocephaloidea.

Scolex armed or unarmed. Two groove-like suckers. Three genital orifices. Eggs like those of *Fasciolidea*, but not always with a lid.

(*a*) Suctorial tubes or cephalic sucker. Uterus rosette-shaped. Eggs with lids.

Genera:—*Ligula.*
Schistocephalus.
Dibothriocephalus.
Diplogonoporus.
Diplocotyle.
Cyathocephalus.
Bothrimonus.
Fistulicola.
Ancistrocephalus.
Triænophorus.

(*b*) Scolex unarmed. Uterus not rosette-shaped. Egg with thin shell and no lid.

Genera:—*Amphicotyle.*
Abothrium.
Ptychobothrium.

(*c*) Vitellogene gland in medullary layer. Uterus with cavity. Eggs with thin shells and no lid.

Genus :—*Amphitretus.*

II. Tetraphyllidea.

Scolex armed or unarmed. Four motile bothridia or four round suckers. Segmentation always distinct. No uterine orifice. Cirrhus and vagina open at the border. Eggs thin-shelled, with no lid.

(*a*) Hooks in the bothridia. Also accessory suckers.

Genera :—*Onchobothrius.*
Calliobothrium.

(*b*) Bothridia without hooks. Accessory suckers not constant.

Genera :—*Anthobothrium.*
Phyllobothrium.

(*c*) With four suckers, mostly unarmed.

Genus :—*Ichthyotænia.*

III. Cyclophyllidea.

Scolex with four suckers. Apical rostellum occasional. Hooks on rostellum; rarely on suckers. Segmentation generally distinct. No uterine orifice. Vagina usually opening at border. Genitalia rarely duplicated. Vitellogene gland single; usually behind ovary. Eggs thin-shelled, without lids. Oncospheres with one or several integuments.

(*a*) Genital pores on the flat surface.

Genus :—*Mesocestoides.*

(*b*) Without vagina.

Genus :—*Acolëus.*

(*c*) Vagina on surface. Cirrhus at border.

Genus :—*Amabilia.*

(*d*) Vitellarium in front of ovary. Suckers with muscular process. Genital pores at one side.

Genus :—*Tetrabothrius.*

(*e*) Scolex unarmed. Large. No neck. Uterus oblique. Eggs with "pyriform apparatus."

Genera :—*Anoplocephala.*
Bertia.
Stilesia.

(*f*) Rostellum armed. Suckers unarmed. Genital pores marginal. Genitalia simple or duplicated. Uterus becoming atrophied. Eggs then free in parenchyma.

Genera :—*Dipylidium.*
Cotugnia.
Hymenolepis.
Dilepis.

(*g*) Rostellum and suckers armed. Eggs mostly encapsuled.

Genus :—*Davainea.*

(*h*) With rostellum. Usually a double crown of hooks. Uterus with median trunk and lateral branches.

Genus :—*Tænia.*

IV. Echinobothriidæ.

Scolex with head and neck. Head with rostellum and two bothridia. Neck with longitudinal row of T-shaped hooks. Genital pores on the flat surface.

Genus :—*Echinobothrium.*

V. Rhynchobothriidæ.

Scolex with head and neck. Head with two or four suckers. Four retractile and armed rostella. Neck unarmed.

Genus :—*Rhynchobothrius.*

Notes on the Chief Cestodes of Man (after Braun)—
ibothriocephalus latus (L.), 1748 (the largest human tape-worm).—
ngth 2 to 9 metres, sometimes 16. Head almond-shaped, 2 to
mm. long. Proglottides may number 4,000 or more, and are
eater in breadth than length.
The testes are situated dorsally and laterally in the medullary
er.
The genital pore is placed centrally.
The eggs (Fig. 4) are large (0·07 by 0·045 mm.), with brownish
ells and small lids. They are deposited in the intestine, and pass
with the fæces.
The embryos hatch out in water after several weeks, the onco-
here being provided with cilia.
The method of invasion of the intermediate host is yet unknown.
The cysticercus stage is passed in the intestinal wall, the liver,
spleen, or the muscular system of various fresh-water fishes,
h as the pike and perch.

The formation of proglottides in the ultimate host may attain a number of 30 or more each day. The dog, and occasionally the cat or fox, may act as an ultimate host as well as man.

The geographical distribution is fairly wide in Europe.

It also occurs in Japan, Turkestan, and Central Africa, and, probably, elsewhere.

The clinical disturbances are generally trifling, and consist of slight gastric or nervous disorders, and occasionally anæmia.

Dibothriocephalus cordatus (R. Lenck), 1863.—Length 80 to 155 centimetres. Head heart-shaped, 2 by 2 mm. Suctorial grooves on the flat surface. The proglottides may number 600. Uterus rosette-shaped. Eggs (0·075 by 0·05 mm.) have lids.

This worm is a common parasite of the seal, walrus, and dog in Greenland and Iceland, and occasionally of man.

The cysticercus stage is probably passed in fishes.

It has not yet been recorded from the tropics.

Diplogonoporus grandis (R. Blanch) 1894, (formerly called *Krabbea grandis*).—Scolex unknown. Chain of proglottides 10 metres long. They are short, but broad.

There are two longitudinal grooves on the vertical surface, in which lie the genital pores.

The ova are thick shelled and brown (0·063 by 0·05 mm.).

It has twice been observed in Japanese; and allied species are found in whales and seals.

Bothriocephalus mansoni (Cobb.), 1883.—The adult stage is unknown; and nothing is known of its origin or development.

Cysticercus forms were discovered by *Manson* in Amoy in 1882, during the post mortem on a Chinaman. Twelve were found beneath the peritoneum and one free in the abdomen. They can migrate and even reach the urinary passages or form excrescences on the skin.

Dipylidium caninum (L.), 1758 (formerly called *Tænia canina*).—The worm is 15 to 35 centimetres in length by 1·5 to 3 mm. in breadth.

The scolex is small and rhomboidal. There is a club-shaped rostellum bearing 3 or 4 rings with some 60 thorn-like hooks.

The segments are longer than wide; and of a reddish colour.

The genital pores are lateral and symmetrical.

The eggs are globular (0·043 to 0·05 mm.).

It is a common intestinal parasite of dogs.

Twenty-three cases have been reported in children and one in an adult.

The cysticercus form lives in the dog-louse and the dog-flea.

Hymenolepis nana (v. Sieb.), 1852 (formerly called *Tænia nana*).—This is the smallest human tape-worm. It is 10 to 15 mm. long by 0·5 mm. broad. The rostellum has a crown of 30 hooks (Fig. 5).

[To face p. 7[illegible]]

Fig. 4.—Ovum of *Dibothriocephalus latu*

Fig. 5.—*Hymenolepis nana.*

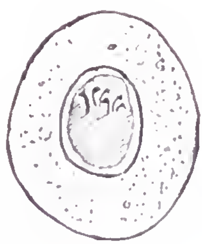

Fig. 6.—Ovum of *Hymenolepis nana* (× 500).

The proglottides number 150 and are short and broad.

The eggs (Fig. 6) are oval—0·035 by 0·05 mm.

It was first discovered, in a boy in Cairo, by *Bilharz* in 1851.

Since 1885 numerous cases have been reported from Italy, Russia, England, France, Germany, S. America, Siam, and Japan.

It produces marked symptoms in children.

The development and method of infection are unknown.

Possibly insects and myriapoda may act as intermediate hosts.

Hymenolepis diminuta (Rad.), 1819.—This tape-worm measures 20 to 60 centimetres in length by 3·5 mm. in breadth.

The proglottides may number 1,000.

The head is very small and club-shaped, and has a rudimentary unarmed rostellum.

The segments measure 3·5 mm. in breadth by 0·66 mm. in length.

The eggs are oval (0·065 by 0·08 mm.), and are thick and yellowish.

The normal ultimate hosts are rats and mice, but it is occasionally found in man.

The cysticercus stage is passed in a small moth (*Asopia farinalis*) as well as in some coleoptera.

Hymenolepis lanceolata (Block), 1782.—The length is about 100 mm. and the breadth 5 to 18 mm.

The head is very small and globular.

The rostellum is crowned with eight hooks.

The genital pore is lateral. The eggs are oval and wrinkled, and measure 0·05 by 0·035 mm.

The normal ultimate hosts are duck, geese, and water fowl.

The intermediate host is the fresh-water cyclops.

It has been recorded, in man, from England, France, Denmark, Austria, and Germany, but, so far, not from the tropics.

Davainea Madagascariensis (Davaine), 1869.—The measurement 25 to 30 cm.

The head has four suckers. The rostellum has 90 hooks.

There are about 600 proglottides, which are 2 mm. long by 1·4 mm. broad.

The eggs lie free in the parenchyma; they are globular, and measure 0·008 mm.

The adult has, so far, only been found in man, in Mauritius, Bangkok, and British Guiana.

The intermediate host is unknown.

Tænia solium (L.), 1767.—The length is usually from 2 to 3 metres or more. The head is globular. There is a short rostellum (Fig. 7), with 26 or 28 hooklets in a double row, large and small alternating. The rostellum may be pigmented.

The suckers are four in number, and are hemispherical.

The neck is thin, and has a length of 5 to 10 mm.

The number of proglottides (Fig. 8) averages 850. They are about 11 mm. long by 5 mm. broad.

The genital pores are lateral and alternate.

The uterus is branched. The ova (Fig. 9) are globular (diameter 0·035 mm.).

The oncospheres have six hooks.

As an adult the worm is found exclusively in the small intestine of man.

The cysticercus stage is usually passed in the muscular tissue of the pig, but may occasionally be found in the stag, wild pig, dog, cat, brown bear, monkey, and man.

The geographical distribution corresponds with that of the domestic pig. The prevalence of the Mohammedan faith will account for the infrequency of human infection in the East.

The infection of man with cysticerci can only occur by the ingestion of the oncospheres with impure water or unwashed vegetables. The possibility of contamination of such foodstuffs is rare in civilised countries, consequently a cysticercal infection is but seldom met with.

Tænia saginata (Goeze), 1782.—The length of this tape-worm averages 8 metres, and may extend up to 36 metres.

It is the commonest of all human tape-worms and has a world-wide distribution.

The head is cubical (diameter 2 mm.) There are four pigmented hemispherical suckers (Fig. 10).

Fig. 7.—Scolex of *Tænia solium.*

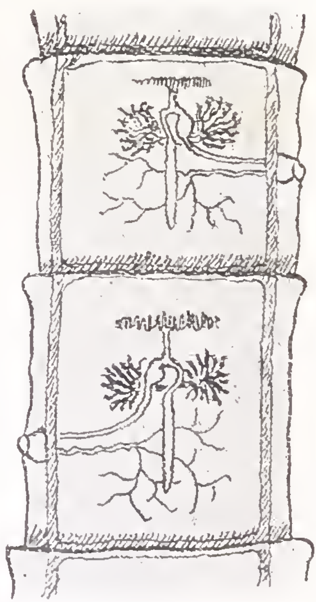

Fig. 8.—Proglottides of *Tænia solium*

A pseudo-sucker takes the place of the rostellum.

Facing p. 7

Fig. 9. Ovum of *Tænia solium* (× [illegible])

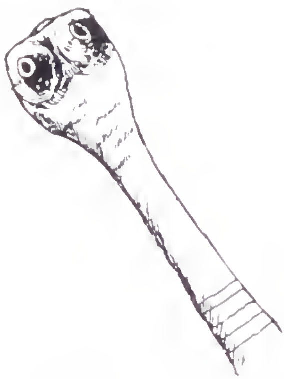

Fig. 10. Scolex of *Tænia saginata.*

Fig. 11. Ovum of *Tænia saginata.*

There is a moderately long neck, half the breadth of the head.

The average number of proglottides is more than 1,000, and, when mature, measure 16 to 20 mm. in length by 4 to 7 mm. in breadth.

The genital pores are lateral and irregularly alternate.

The uterus is branched. The ova are transparent and oval, measuring 0·04 mm. by 0·03 mm. (Fig. 11).

Fig. 12.—Proglottides of *Tænia confusa*

The adult is exclusively an intestinal parasite of man.

The cysticercus form is almost as exclusively found in the ox. Human cysticercal infection with this species is very rare.

Tænia Africana (v. Lstw.), 1900.—The adult worm is about 1·5 metres in length.

The scolex is unarmed and has an apical sucker.

The neck is short and broader than the scolex.

The average number of proglottides is about 600, and they measure 7 mm. in length by 12 to 15 mm. in breadth.

The genital pores are lateral and irregularly alternate.

There is a non-branching double uterus.

The ovum is round (0·032 mm.) or oval, and has radial stripes on the shell.

Tænia confusa (Ward), 1896.—Only two specimens have been recorded, both in human beings, in America.

The length is 8·5 metres.

The scolex is unknown.

The proglottides number 800, being (when mature) 35 mm. long by 4 to 5 mm. broad (Fig. 12).

The genital pores are lateral and irregularly alternate.

The uterus is ramified. The ova are radially striated, oval in shape, and measure 0·039 by 0·3 mm.

Tænia echinococcus (v. Sieb.), 1853.—This small tape-worm measures 5 to 6 mm. in length (Fig. 13).

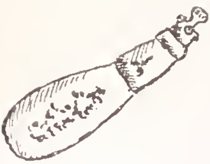

Fig. 13.—Scolex of *Tænia echinococcus*.

There is a rostellum with a double row of 28 to 50 hooklets.

There are four round suckers. The neck is short.

There are only three or four proglottides, the distal one being 2 mm. by 0·6 mm.

The genital pores alternate.

The uterus is tubular, with lateral protuberances. The ovum is globular (0·035 mm.).

The normal host of the adult is the dog, but it is also found in the jackal and wolf. Its habitat is the small intestine, and considerable numbers of parasites are usually present in one host.

The cysticercus stage is usually passed in the liver, lungs, or other organs of some 27 different mammals, chiefly sheep, oxen, and pigs.

Man is not uncommonly affected with this cysticercus, probably always contracted by the infected carcsses of dogs.

The growth of the encysted parasite is slow, and, as a rule, five months or more will elapse before the first brood capsules with scolices are formed.

The distribution of human cysticercal infection includes Iceland, Europe, Australia, and many parts of Africa.

TREMATODES (Rudolphi), 1809.—These "sucking worms" or "flukes" are usually leaf- or tongue-shaped and seldom cylindrical.

CESTODES OF MAN.

Name.	Geographical Distribution.	Intermediate Host.	Ultimate Host.	Length.	Number of Segments.	Characters of Scolex.	Proglottis (mm.)		Genital Pore.	Uterus.	Ova (in mm.).
							Length.	Breadth.			
Dibothriocephalus latus.	Europe, Japan, Turkestan, Africa.	Fresh-water fish.	Dog. —— Man.	Up to 16 metres.	4,000	Two longitudinal suckers.	5	12	Ventral.	Rosette in centre of proglottis.	Large, oval, operculated, 0·07 × 0·045.
Dibothriocephalus cordatus.	Iceland and Greenland.	? Fish.	Seal, walrus, and dog. —— Man.	Up to 115 centimetres.	600	Two suctorial grooves.	8	4	Ventral.	Rosette shaped.	Operculated, 0·075 × 0·05.
Diplogonoporus grandis.	Japan.	? Fish.	Whales, seals. —— Man.	10 metres.	3,000	(Unknown).	0·45	25	Ventral.	Branching.	Operculated, 0·063 × 0·048.
Bothriocephalus mansoni.	China.	(Subperitoneal cysticercus forms discovered in Amoy in a Chinaman).									
Dipylidium caninum.	Europe and America.	Dog-lice and dog-fleas.	Dogs. —— Man.	35 centimetres.	200	Rostellum with 60 hooks and 4 suckers.	7	3	Lateral and symmetrical.	Single and central.	0·043 × 0·05.
Hymenolepis nana.	Egypt, Europe, S. America, Siam, Japan.	Myriapoda.	Man.	15 millimetres.	150	Four suckers.	7	12	Lateral and irregularly alternate.	Radiating diverticula.	Oval, 0·035 × 0·05.
Hymenolepis diminuta.	America and Europe.	Moths and coleoptera.	Rats and mice. —— Man.	60 centimetres.	1,000	Four suckers.	0·66	3·5	Lateral.	?	Oval, thick, and yellow, 0·065 × 0·08.
Hymenolepis lanceolata.	Europe.	Cyclops.	Ducks, geese. —— Man.	100 millimetres.	...	Rostellum with 8 hooks.	...	...	Lateral.	...	Oval and wrinkled, 0·05 × 0·035.
Davainea Madagascariensis.	Bangkok, Mauritius, British Guiana.	? (Cockroach?)	Man.	30 centimetres.	600	Four suckers and rostellum with 90 hooks.	2	1·4	Lateral and same side.	Transverse rows of balls.	Globular, 0·008.
Tænia solium.	Corresponds with that of domestic pig.	Pig, dog, monkey. —— Man.	Man.	3 metres.	850	Four suckers and rostellum with 28 hooks.	11	5·5	Lateral and alternate.	Branched.	Globular, 0·035.
Tænia saginata.	Ubiquitous.	Ox.	Man.	8 (? to 36) metres.	1,000	Four suckers.	18	6	Lateral and irregularly alternate.	Branches.	Transparent and oval, 0·04 × 0·03.
Tænia Africana.	Central Africa.	Zebu.	Man.	1·5 metres.	600	Apical sucker.	7	14	Lateral and irregularly alternate.	Double and non-branching.	Round, 0·032.
Tænia confusa.	America.	?	Man.	8·5 metres.	800	(Unknown).	35	5	Lateral and irregulary alternate.	Ramified.	Oval, 0·039 × 0·3.
Tænia echinococcus.	Iceland, Europe, Australia, and Africa.	Sheep, oxen, pigs, &c. —— Man.	Dog.	6 millimetres.	3	Four suckers and rostellum with 50 hooks.	2	0·6	Lateral and alternate.	Tubular with lateral protuberances.	Globular, 0·035.

They live as ecto- or endo-parasites, and are only ciliated in the arval condition. The adults are parasites on vertebrate animals, he intermediate generation being passed in molluscs.

The worms are generally hermaphroditic.

The surface on which the genital openings occur is called the entral surface.

The oral aperture is always at the anterior end, and acts as an nus, except in *Gasterostomium*—a parasite of fishes.

Sucking discs are common, and have chitinous hooks or claspers n their vicinity.

Anatomy. The body is covered with a chitinous *cuticle*, conaining unicellular glands.

The *parenchyma* is a connective tissue consisting of branching nd anastomosing multipolar cells.

The *muscular system* consists of a musculo-dermal tube, some arenchymal muscles, and fibres attached to the suckers, hooks, nd spines.

The *nervous system* is composed of two anterior ganglia connected y a commissure. From these, three fasciculi proceed forward, on ach side, to the sucker; and three proceed backwards, being nited by a number of transverse commissures.

The peripheral nerves either spring from these posterior nerve unks or from the commissures. Some of them pass directly to the uscles; others form plexuses.

Sense Organs.—Two or four simple eyes occur in some ectoarasitic species, and in a few endo-parasitic forms while in the ee larval stage.

The Alimentary System.—There is a sucker with an oral cavity, n œsophagus, and a muscular pharynx.

The intestine divides into two lateral cæca.

(In some species there is a connection between one of these ranches and the genital system.)

The anterior alimentary cavities are lined with a reflexion of the ternal skin, but the intestine with cylindrical epithelium.

Salivary glands discharge into the œsophagus in the neighbourod of the pharynx.

The *food* of the trematodes consists of mucus, cells, blood, or bris. Such of this as is soluble is distributed through the renchyma, and excreted by a symmetrical water-vascular system ich opens into an excretory pore—the foramen caudale—in the ddle of the posterior border. If there is a sucker present, e excretory pore is placed in front of the sucker on the dorsal face.

The contents of the system consist of a clear or reddish fluid.

Sexual System.—Most of the trematodes are hermaphrodites. A v, such as the *Schistosoma*, are sexually differentiated.

The *male organs* consist of two testes with their vasa efferentia iting to form a muscular vas deferens which opens close to, or ectly into, the genital pore.

In the atrium is a muscular cirrus pouch which may be projected, and serves as an organ of copulation.

The *female organs* include an ovary; a pair of vitelline sacs; a short oviduct; Laurer's canal (a tube originating on the dorsal surface, and having a receptaculum seminis suspended to the inner end); and a uterus.

The fertilised cells with their shell material and vitellus pass to the uterus which occupies a considerable part of the central field. Its terminal part lies next to the cirrus pouch, and discharges near the male orifice either into a genital atrium or on to the body surface.

This terminal portion of the uterus serves as a vulva—*Metraterm.*

Life History.—The ova, being deposited, leave the affected organs by natural channels. When they have reached water, as a rule, the embryos will hatch out; in other cases it is necessary that they be ingested by some intermediate host, and are only hatched under the influence of intestinal juices.

The ciliated embryos—known as *Miracidia*—sooner or later penetrate the body of an intermediate host—a fresh-water mollusc—and become a germinal body, without an intestine, called a *Sporocyst.* By another intermediate generation they become what is known as a *Redia*, a somewhat similar body to a sporocyst, but possessing an alimentary canal.

A further intermediate generation in a fresh host results in their becoming an adult known as a *Cercaria.* These adults leave their host, and are able to move in the water by means of a rudder-like tail.

A final entry into another molluscan host is made; where the tail is lost and they become encysted. Other species of cercaria may encyst themselves in the water or on plants.

In either case they are taken up by an ultimate host, such as man or the sheep, &c., in which they develop and reach maturity.

Endo-parasitic trematodes usually only occur in vertebrate animals, and they may inhabit almost any organ. The intestine is the favourite site, then the liver, bile ducts, or gall bladder.

Classification (after Braun).

I. Heterocotylea.

Exclusively ecto-parasitic. Well developed clasping organs. Excretion on dorsal surface at anterior end. Live chiefly on gills of fish, in the bladder of amphibians, and œsophagus of tortoises.

II. Aspidocotylea.

Endo-parasitic. Low organisation. Large ventral sucker. Excretion by posterior pore. Parasitic in intestine of fish, and tortoises.

III. Malacocotylea.

One or two sucking discs. Oral orifice anterior. Intestine forked. Genital pore on ventral surface. Excretion at posterior end. Sexual organs usually combined. Endo-parasitic in vertebrates.

(1) Development without alternation of generations, but with formation of two larval forms and a change of hosts.

Genera :—*Hemistomum.*
Holostomum.
Diplostomum.
Polycotyle.

(2) Development complicated by interpolation of generations that reproduce asexually (sporocysts, redia, &c.), and by one or two changes of host.

(*a*) Ventral sucker at posterior end. Excretion on dorsum. Genital pore median line of ventral surface. Intestine without appendages. Pharynx anterior and termed "oral sucker." All hermaphroditic.

Genera :—*Paramphistomum.*
Gastrothylax.
Gastrodiscus.

(*b*) With oral and ventral suckers. Excretion at posterior border. Genital pore on ventral surface or lateral border. Intestinal fork without appendages. All hermaphroditic.

Genera :—*Fasciola.*
Fasciolopsis.
Paragonimus.
Opisthorchis.
Cotylogonimus.
Dicrocœlium.

(*c*) Structure as in (*b*), but sexes distinct.

Genera :—*Schistosomum.*
Bilharziella.

(*d*) Sexually distinct. Occur in pairs. Are encysted. Found in fish.

Genera :—*Didymozoon.*
Nematobothrium.

(*e*) Structure as in (*b*). Oral and ventral sucker. Two retractile rostrella. Genital pore ventral and anterior. Genital glands posterior.

Genus :—*Rhopalias.*

(For notes on the Human Trematodes see Chapter xiv. on Bilharziasis and Chapter xix. on Distomiasis.)

NEMATODES (Rudolphi), 1809.—Nematodes are elongated filiform or fusiform worms with a chitinous cuticle either smooth or annulated.

They have an alimentary system and an anus.

The sexes are usually separate; the genitalia of the male open into the anus, those of the female in the median ventral ridge about the middle or towards the anterior end of the body.

Some live non-parasitically in water, soil, &c.; others are parasitic in animal organs or in plants.

Anatomy.—There is either a thiek or thin chitinous eutiele, in whieh open unicellular eutaneous glands.

The cutis is approximated to the dermo-museular tube which contains a single layer of longitudinal muscle fibres which ean shorten or bend the body.

There are some four, six, or more ramified eells, up to 1 em. in size, whieh lie elose to the walls of the body cavity, and are ealled "phagoeytic organs."

In *Ascaris* they are to be found only at the anterior end of the body; in other genera, sueh as *Strongylus*, they may extend the whole length of the body, eorresponding in position to the lateral ridges.

The Alimentary System.—There is an oral cavity frequently surrounded at its aperture by bristles or papillæ.

The œsophagus is a short, muscular, bottle-shaped tube lined by a prolongation of the external body cutiele.

There is a bulb-like dilatation of the posterior part of the œsophagus—the stomaeh.

From this is continued the mid-gut, a flattened tube lined with cylindrical or flat cells. Intestinal eæea are sometimes present.

There is a short eetodermal hind-gut.

The Nervous System contains a eentral nervous organ composed of a fascicular ring (around the œsophagus) remarkable for the number of its fibres and ganglion cells.

From this ring, a *ventral median nerve* proceeds backwards, and likewise a *dorsal median nerve*, both of whieh are connected by semicircular eommissures asymmetrically placed.

Towards the posterior part of the body there is an anal ganglion on the ventral median nerve, and a faseieular ring conneeting it with the dorsal median nerve.

The two median nerves are ehiefly motor.

Two rudimentary eyes are sometimes present in the non-parasitic genera. In the parasitie forms they are absent.

Sensory fibres supply the oral papillæ.

The Excretory System.—In some eases this consists of symmetrical tubes running in the lateral ridges, uniting at the anterior extremity into a short duct opening into an excretory pore.

In other cases the right braneh may be absent.

Connected with the excretory duct is a horse-shoe-shaped cell with an intracellular tubular system.

In a few genera, a special excretory system is absent, its place being taken by the cutaneous glands.

The Generative System.—In the *male* there is a single straight or sinuous testis passing into the spermatic duct. This duct contains a dilated vesiculus seminalis and a muscular ejaculatory duct which opens into the hind-gut.

The spermatozoa, which are amœboid but have no flagella, only attain their full development in the uterus.

The *female* organs consist of an orifice—the vulva—situated ventrally and at about the middle of the body: a short vagina terminating in a bi-lobed uterus, contiguous to which are two long filiform ovaries.

Life History.—Fertilisation takes place in the uterus. A few genera are viviparous, but in most nematodes the eggs are passed and are subsequently developed in moist earth or water.

Thick-shelled eggs can resist prolonged desiccation.

In the non-parasitic forms the embryos are miniature adults after hatching out. In most of the parasitical forms they are really larvæ.

Ascaris and *Trichocephalus* will develop if the ova are swallowed by a suitable host.

In *Strongylus* and other genera, the larvæ are hatched and live and moult in water, &c., and gain their host by his ingestion of the vehicle in which they are contained.

Sometimes, however, the nematode larvæ make use of one or more intermediate hosts; but there is no multiplication in them as in the corresponding phase of cestodes and trematodes.

A cat nematode—*Ollulanus tricuspis*—passes its encysted larval stage in the muscles of mice.

In *Trichinella spiralis*, which inhabits the intestines of some mammals and occasionally man, there is viviparous reproduction. The same host, however, is used by the larvæ who reach the muscles *via* the lymph stream and blood.

Classification (after Braun).—This is not very satisfactory. Nematodes are usually divided into a number of families.

Fam. 1. *Enoplidæ.*

A non-parasitic marine family.

Fam. 2. *Anguillulidæ.*

Live free in fresh water or soil.
A few are parasitic.
Œsophagus has a double dilatation.
Males have two spicules.
Females have pointed tails.
Vulva is in middle of body.

Genera:—*Anguillula.*
Rhabditis.
Heterodera.

Fam. 3. *Angiostomidæ.*

Characterised by heterogony—*i.e.*, alternation of two differently-structured sexual generations.

Genera:—*Rhabdonema.*
Angiostoma.
Allantonema.

Fam. 4. *Gnathostomidæ.*

Ramified spines covering whole or part of body.
Stomach parasite of vertebrates.

Genus:—*Gnathostoma.*

Fam. 5. *Filariidæ.*

Filiform nematodes.
Oral aperture often surrounded by papillæ.
Œsophagus slender and without bulb.
Male has one or two uneven spicules.
Vulva is anterior.
Generally ovoviviparous.

Genera:—*Filaria.*
Spiroptera.
Dispharagus.

Fam. 6. *Trichotrachelidæ.*

Œsophagus resembles a pearl necklace.
Anterior of body slender.
Spicules one or none.
Ovary single.
Vulva central.

Genera:—*Trichinella.*
Trichocephalus.
Trichosoma.

Fam. 7. *Strongylidæ.*

Possess six oral papillæ.
Male has a bursa copulatrix.
Individuals are small.
Family is a large one.

Genera:—*Eustrongylus.*
Strongylus.
Syngamus.
Sclerostoma.
Ankylostoma.
Dochmius.

Fam. 8. *Ascaridæ.*

Mouth with three papillæ.
Œsophagus with bulb.
One or two spicules.
Ovary double.

Genera:—*Ascaris.*
Oxyuris.
Heterakis.

Notes on the Chief Nematodes of Man* (after Braun)—
Rhabditis pellio (Schneider), 1866.—The males are 0·8 to 1·05 mm. long; the females, 0·9 to 1·3 mm.

The posterior end of the male has a heart-shaped bursa with 7 to 10 ribs on each side.

There are two uneven spicules.

The female posterior is long and pointed.

The ovary is single.

The vulva is behind the middle of the body.

The eggs are oval, 0·06 to 0·035 mm.

The larval stage is parasitic in earth-worms. The adult lives in decomposing matter in the soil, but is a facultative parasite and has been known to multiply in the human vagina.

Strongyloides intestinalis (Bavay), 1877.—This nematode is found in China, the West Indies, Brazil, Africa, and Europe.

Generation is by heterogony, or alternation of reproduction.

One generation is *parasitic* (*Anguillula intestinalis*). Length, 2·2 mm.; breadth, 0·034 mm.

The cuticle is transversely striated.

The mouth has four lips.

The œsophagus is long and cylindrical.

The anus opens in front of the posterior extremity.

The genital orifice is in the posterior third of the body.

Reproduction is hermaphroditic.

It is introduced into its human or other host by food or water. Reaching the intestine it bores into the mucous membrane; and in that situation the eggs (which measure 0·055 by 0·032 mm.) are developed. When hatched out the young (measuring 0·2 mm.) again reach the lumen of the bowel and largely increase in size, often causing diarrhœa, until at last they are voided with the fæces.

They then become a *free-living* generation (*Anguillula stercoralis*) which is sexually differentiated.

The body is smooth, cylindrical, and slender in front.

The mouth has four indistinct lips.

The œsophagus is short with a double dilatation.

The opening of the anus is in front of the tail.

The males are 0·7 mm. in length, and carry the posterior end rolled up.

* See also Chapter xii., on *Ankylostomiasis*; and Chapter xxii., on *Filariasis*.

The females measure 1 mm. in length, and the tail end is straight and pointed.

The vulva is rather behind the middle of the body.

In about thirty hours after leaving the body of their parents' host they copulate, and the female deposits 30 to 40 eggs (oval, 0·07 by 0·045 mm.) which rapidly develop.

The resulting embryos exhibit the features of their parents (rhabditis form). They continue to grow and soon moult, losing their own characteristics and acquiring those of their grandparents (strongyloid form).

They then die off unless they reach the intestine of a suitable host, and so restart the double cycle of generations.

Trichocephalus dispar (Rud.), 1801.—This is also known as *T. trichiurus*, and is the common "whip worm." It is one of the commonest of the tropical parasites of man, and has also been found in monkeys and lemurs.

The male is 40 to 45 mm. in length, about half of which is filiform.

There is a spiculum 2·5 mm. long, in a spinous retractile pouch.

The female measures 45 to 50 mm., of which length $\frac{2}{5}$ are filiform.

The ova measure 0·05 by 0·023 mm., are brown, barrel-shaped, and perforated at the poles (Fig. 14).

The development of the eggs takes place in water or in moist soil.

Trichinella spiralis (Owen), 1835.—This nematode, as an adult, is a parasite of various mammals, of which the small intestine is the usual habitat.

The known hosts are man, the black rat, the sewer rat, domestic and wild pigs, the dog, the fox, the badger, and the cat—the latter four being least easily infected.

The male is 1·5 mm. in length and 0·04 mm. in diameter.

The anterior part of the body is narrow; the cloaca is terminal, and lies between the two caudal appendages.

The female is 3 to 4 mm. in length and 0·06 mm. in diameter.

The anus is terminal. Copulation takes place in the duodenum and jejunum, after which the males die off.

The impregnated females burrow into the mucous membrane and reach the lymphatic spaces, where they deposit larvæ to the number of some 1,500. Their size is 0·1 by 0·006 mm., and their migration is a passive one, brought about by the lymph stream and circulating blood. In this way muscular tissue throughout the whole body may be invaded.

It requires some ten days after infection for the embryos to reach their destination. The affected muscle fibre degenerates and becomes inflamed. In their encysted condition the embryos may remain alive for eleven years in the pig and twenty-five to thirty years in man.

The normal hosts are the black and brown rats.

The geographical distribution of the nematode is almost world-wide, but its occurrence amongst human beings is more limited, owing to religious prejudices against the flesh of the pig.

Eustrongylus gigas (Rudolphi), 1802.—A blood-red nematode, parasitic in the kidney of the dog, seal, horse, &c., but known occasionally in human beings.

There are about 150 papillæ along the lateral ridges.

The males are 40 centimetres in length by 5 mm. in diameter.

The anal orifice is in a collar-like bursa armed with papillæ; and the spicule is 5 mm. long.

The females are 100 centimetres long by 12 mm. in diameter.

The anus is terminal and of a crescentic shape.

The vulva is 60 mm. from the anterior end.

The eggs are oval and brownish, and have a thick shell (with numerous depressions) measuring 0·064 by 0·04 mm.

The eggs develop in water or moist soil. An intermediate stage in fishes is possible.

Strongylus apri (Gmelin), 1789.—This nematode is a parasite inhabiting the bronchial tubes of the pig and wild boar, but has been occasionally found in man and the sheep.

No intermediate host is necessary.

The male is 12 to 25 mm. long. The bursa copulatrix is two-lobed.

The spicules are thin and have a length of 4 mm.

The females are 50 mm. long, with a recurved anus just in front of the posterior extremity.

The eggs are eliptical and measure 0·1 by 0·05 mm.

When laid, the embryo is already formed within.

Strongylus subtilis (Looss), 1895.—This parasite has been found in the human small intestine in Egypt, and in the stomach of a Japanese woman. It is also a parasite of the camel.

The male is 4 to 5 mm. long, and is considerably more slender in front than behind.

There is a large muscular œsophagus.

The spicules are equal and attain a length of 0·15 mm., having some supports springing from the front.

The female is about 6 mm. in length.

The anus is just in front of the posterior extremity, which tapers into a slender tail.

The vulva is at the last fifth of the body.

The ova are thin-shelled and oval (0·063 by 0·041 mm.), and are deposited before segmentation.

Ascaris lumbricoides (L.), 1758.—A red or greyish-yellow worm, known to the Greeks as ἕλμινς στρογγύλη, and to the Latins as *Tinea rotunda*.

NEMATODES OF MAN.

(*For Ankylostomes see Chapter xii.; for Filariæ see Chapter xxii.*)

Name.	Geographical Distribution.	Situation in Man.	Length of Adult (in mm.).		Size of Ova (in mm.).	Development of Embryos.
			Male.	Female.		
Rhabditis pellio.	Hungary.	Facultative parasite of vagina.	0·8 to 1·05	0·9 to 1·3	Oval, 0·06 × 0·035.	Parasitie in earth worms.
Strongyloides intestinalis.	China, W. Indies, Brazil, Africa, and Europe.	Parasite of intestine (hermaphrodite).	...	...	Oval, 0·055 × 0·032 (are hatched in m. membrane of intestine).	Free living generation. Males, 0·7 mm.; females, 1 mm.
Trichocephalus dispar.	Widespread.	Cæcum.	40 to 45	45 to 50	Oval, with perforated poles, 0·05 × 0·023.	In water or moist soil.
Trichinella spiralis.	Widespread, but human infection limited *qua* use of pigs' flesh.	Small intestine. *N.B.*—The normal host is the rat.	1·5	3 to 4	0·1 × 0·006.	Larvæ deposited in the mueous membrane, and work their way to muscles, where they become eneysted.

Eustrongylus gigas.	Europe, N. and S. America.	Kidney (normal hosts = dog, seal, horse, &c.).	40 cm.	100 cm.	Oval, brown, with depressions, 0·064 × 0·04.	Eggs develop in water or moist soil. Intermediate stage in fish is possible.
Strongylus apri.	Transylvania.	Bronchial tubes (normal hosts = pig and wild boar).	12 to 25	50	Elliptical, 0·1 × 0·05.	Embryo already formed when eggs are laid.
Strongylus subtilis.	Egypt and Japan.	Small intestine (normal host = camel).	4 to 5	6	Thin-shelled and oval, 0·063 × 0·041.	Ova deposited before segmentation. Develop in water or moist soil.
Ascaris lumbricoides.	Ubiquitous.	Small intestine.	25 cm.	40 cm.	Elliptical and rough, 0·06 × 0·045.	Develop in moist earth or water after long time. Infection is direct.
Ascaris canis.	Widespread.	Small intestine (normal hosts = dog and cat).	50	150	Spherical, thin shell, and rough, 0·068.	Develop in moist earth or water.
Oxyuris vermicularis.	Ubiquitous.	Large intestine.	3 to 5	10	Thin-shelled and oval, 0·05 × 0·02.	Embryo already developed when egg laid. Infection direct.

There are three oral papillæ. Of these the dorsal carries two sense organs, and the two ventral papillæ one sense organ each.

The male is up to 25 centimetres in length by some 3 mm. in diameter.

The curved spicules are 2 mm. long.

The cloacal orifice has 70 to 75 papillæ.

The female may attain a length of 40 centimetres.

The vulva is at the junction of the anterior and middle thirds of the body.

The ova are eliptical, with a thickened, transparent, roughened, and yellow-stained shell. They measure 0·06 mm. by 0·045 mm., and are deposited before segmentation (Fig. 15).

Short periods of freezing and desiccation does not affect the ova. They develop in moist earth or in water after a long incubation.

Direct infection is the general rule, acquired by ingestion of embryo-containing ova.

The small intestine is the normal habitat, but the worms wander considerably.

In geographical distribution it is ubiquitous.

Ascaris canis (Werner), 1782.—This is a common intestinal parasite of the dog and cat. It occurs also in the lynx, lion, &c., and eight cases have been reported in man.

The male is 50 mm. long. The posterior end is bent spirally, and has 26 pairs of papillæ.

The female is 150 mm. long, and the posterior end is straight and conical.

The vulva is in the centre of the anterior end.

The ova are spherical, with a thin shell, and slightly roughened surface. They are 0·068 mm. in diameter.

Oxyuris vermicularis (Linné), 1767.—This parasite was known to the Greeks as ἀσκαρις. It is of ubiquitous distribution, occurs principally in children, and inhabits the large intestine.

Infection is direct and easy.

They are of a white colour, with an annulated cuticle.

There are three retractile oral papillæ.

The male measures 3 to 5 mm. in length, the female 10 mm.

The anus is about 2 mm. in front of the tail extremity.

The vulva is in the anterior third of the body.

The ova are thin-shelled and oval, measuring 0·05 by 0·02 mm. They are laid with embryos already developed (Fig. 16).

The embryo, which has a thin tail, passes into a folded nematode shape, in a short time, if the temperature be sufficiently high.

Auto-infection by the fingers is common.

[To face p. 66.

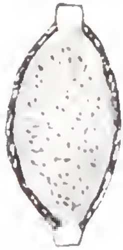

Fig. 14.—Ovum of *Trichocephalus dispar* ([illegible]).

Fig. 15—Ovum of *Ascaris lumbricoides* (× [illegible]).

Fig. 16.—Ovum of *Oxyuris vermicularis* (× 500).

CHAPTER IX.

NOTES ON MOSQUITOES.

CULICIDÆ.

THIS family of the dipterous insects is a highly important and widely distributed one, which causes endless annoyance throughout the tropics generally; and, by its *rôle* of intermediate host in the life history of several important parasites, is a source of great danger to man.

Distribution.—Mosquitoes do not flourish much in temperate climates.

In many parts of the polar circle, as in the North of Canada, Lapland, Greenland, &c., they constitute a veritable pest during the summer months—*Culex pipiens* being the chief form.

In the tropics, however, they are especially numerous and troublesome, a fact partly due doubtless to the climate and partly to the lack of superficial drainage and to the careless domestic habits of the tropical inhabitants.

The various genera are very widely distributed. The two commonest species are (1) *Stegomyia fasciata* (the yellow fever carrier), which may be found almost anywhere between the parallels of 40° N. and S. of the equator, and which is the commonest mosquito of most oceanic islands and seaports within those limits; and (2) *Culex fatigans* (the common brown household mosquito), which has almost the same distribution.

The species of other genera are often especially prevalent in certain continents or districts; while a few genera are entirely confined to certain countries.

The following rough list will show at a glance the main distribution of some of the commoner genera:—

ROUGH TABLE OF THE MORE FAVOURITE DISTRIBUTION OF CERTAIN GENERA.

Temperate climates,	*Culex.* *Grabhamia.* *Œdes.*
Cold zone, . .	*Culex.*
Tropical Hill Districts, .	*Anopheles.* *Theobaldia.*

4. Tropical N. America,
- *Culex.*
- *Grabhamia.*
- *Stegomyia.*
- *Psorophora.* (N. and S. America.)

5. Tropical S. America,
- *Œdes.*
- *Culex.*
- *Mansonia.*
- *Stegomyia.*
- *Psorophora.* (N. and S. America.)
- *Sabethes.* (Only S. America.)
- *Hæmagogus.* (S. America and W. Indies.

6. Tropical Africa, .
- *Anopheles.*
- *Culex.*
- *Mansonia.*

7. Tropical Asia, .
- *Anopheles.*
- *Mansonia.*
- *Stegomyia.*
- *Culex.*

The spread of mosquitoes is both natural and artificial.

The natural diffusion is perforce limited, for it can only be effected by the restricted agency of rivers or winds which can convey larvæ or adults.

The artificial dissemination is accomplished by the agency of ocean-borne transport; and by this means the insects may be carried immense distances, either as hibernating adults or as larvæ in the ship's tanks.

In this way *Culex fatigans* was introduced into Australia, and has since spread inland by the medium of the railways. In all probability it is thus also that *Theobaldia spathipalpis* spread from Europe to the Canaries, the Cape, and to Egypt.

Life History and Habits.—The adult females, after copulation, generally seek water on which to lay their eggs. *Grabhamia*, however, will lay on damp mud; and *Dendromyia Smithii* on leaves.

They alight on a leaf or some floating body, and, supporting themselves on their four anterior legs, cross the posterior legs at an acute angle into which the eggs are dropped, side by side, until an elongated boat-shaped mass is formed, which the insect then allows to drop into the water. Several hundred eggs may be contained in one raft (*Culex* and *Scutomyia*).

Some mosquitoes, however, lay their eggs separately (*Stegomyia*, *Mansonia*, &c.), and others again in long ribbons (*Tæniorhynchus*).

The eggs of the different mosquitoes are often of peculiar shapes (Fig. 17).

The eggs are usually laid betweecn five and six in the morning, and the larvæ escape at periods varying from two to six days or more after being deposited in the water.

The **Larvæ** (Fig. 18) may be siphonate or asiphonate. The latter class is confined to the *Anophelinæ*.

There are several moults, three taking place in the first two or three weeks.

The head has chitinous plates and usually compound eyes.

There are two short antennæ, mandibles, and maxillæ. There is a clypeus and numerous sensitive hairs.

The thorax is large, and has lateral or dorsal bristles.

The abdomen has nine segments, on the eighth of which is the respiratory siphon, in the siphonate larvæ. The ninth segment is short, and has the anus, some plates containing air chambers, and variously arranged bristles. In certain anopheline species, dorsal palmate hairs are found on some of the segments.

Fig. 17.—Culicine Ova.—1, Janthinosoma; 2, Chrysoconops; 3, Mansonia; 4, Stegomyia; 5, Anopheles; 6, Culex.

The characters of specific value in asiphonate larvæ are:—

The frontal hairs.
The palmate hairs.

In siphonate larvæ:

The number of comb spines on the siphon.
The number and form of spines on the basal comb of the eighth segment.

The surface position varies.

In the siphonate forms, the siphon is protruded towards the surface, while the head of the larva depends.

In the asiphonate genera, the larva lies more or less flatly along the surface.

Most larvæ feed on algæ, though some (*Mucidus*, *Toxorhynchus*, &c.) are carnivorous.

The larva eventually places itself horizontally at the surface with the dorsum upwards, and through a rent in the thorax the **Pupa** (Fig. 18) emerges.

This pupa, or nymph, can swim, but takes no nourishment.

The head is folded on the thorax, and has two large black eyes.

Fig. 18.—Larvæ and Pupæ.

The respiratory organs open on the thorax instead of the abdomen, by a pair of siphons through which it obtains air at the surface.

The abdomen has nine segments ending in two broad anal plates.

Specific characters are not marked in the pupa to the same extent as in the larva.

The life span is short, varying from forty-eight hours to a week or more.

When ready to hatch they rise to the surface and straighten themselves out, thus splitting their integument and releasing the imago, who rests for some hours on the floating pupal skin until his wings and body have dried properly.

Both larvæ and pupæ are frequently the prey of fish and aquatic insects.

The larvæ of dragon flies (*Odonata*) and water beetles (*Hydrophilidæ*) devour them with rapidity.

The Imago—Food.—The vast majority of mosquitoes will, of course, never taste human blood, and comparatively few even that

f other mammals or vertebrates. The normal food for all of them probably the juice of leaves, or occasionally fruit. When human lood is available, the following genera are the most blood-thirsty:—

Stegomyia.	*Culex* (some).
Grabhamia.	*Myzorhynchus.*
Mansonia.	*Stethomyia.*

In almost all cases it is the female which bites man, the male eing an exclusive plant-feeder.

It has been erroneously stated that a meal of blood is a necessary reliminary to egg-laying.

Hibernation.—In the tropics mosquitoes continue to breed all ie year, resting during the dry seasons.

In colder climates the females hibernate, while the males die.

The majority of species will hibernate in the adult form.

Anopheles bifurcatus, *Anopheles nigripes*, and *Dendromyia Smithii*

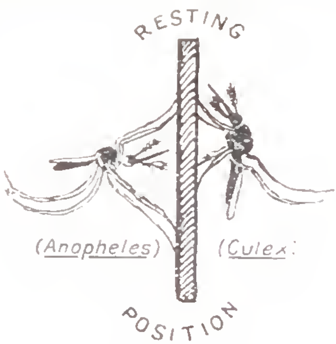

Fig. 19.

rvive the winter as larvæ, and may be frozen and refrozen with ipunity.

Grabhamia dorsalis faces the rigors of winter while still in the um stage.

Activity.—Most mosquitoes are nocturnal feeders, though casionally taking a chance meal in the day time. *Stegomyia sciata*, however, feeds largely during the day as well as night.

The distance over which they are capable of travelling is probably t much over a mile.

Certain species, such as *Stegomyia fasciata* and *Culex fatigans* especially domesticated, and are usually found near human bitations.

The darker mosquitoes (comprising most of the genera) spend day time at rest in dark corners in the house or in the jungle. ightly coloured genera, such as *Megarhininæ* and *Sabatinæ*, seek sunshine.

The resting position of the *Anophelinæ* (except *Myzomyia culicifacies*) rather resembles that of a thorn stuck into the wall. Other mosquitoes do not keep head, thorax, and abdomen in one line, and sit more or less parallel to the surface of support (Fig 19).

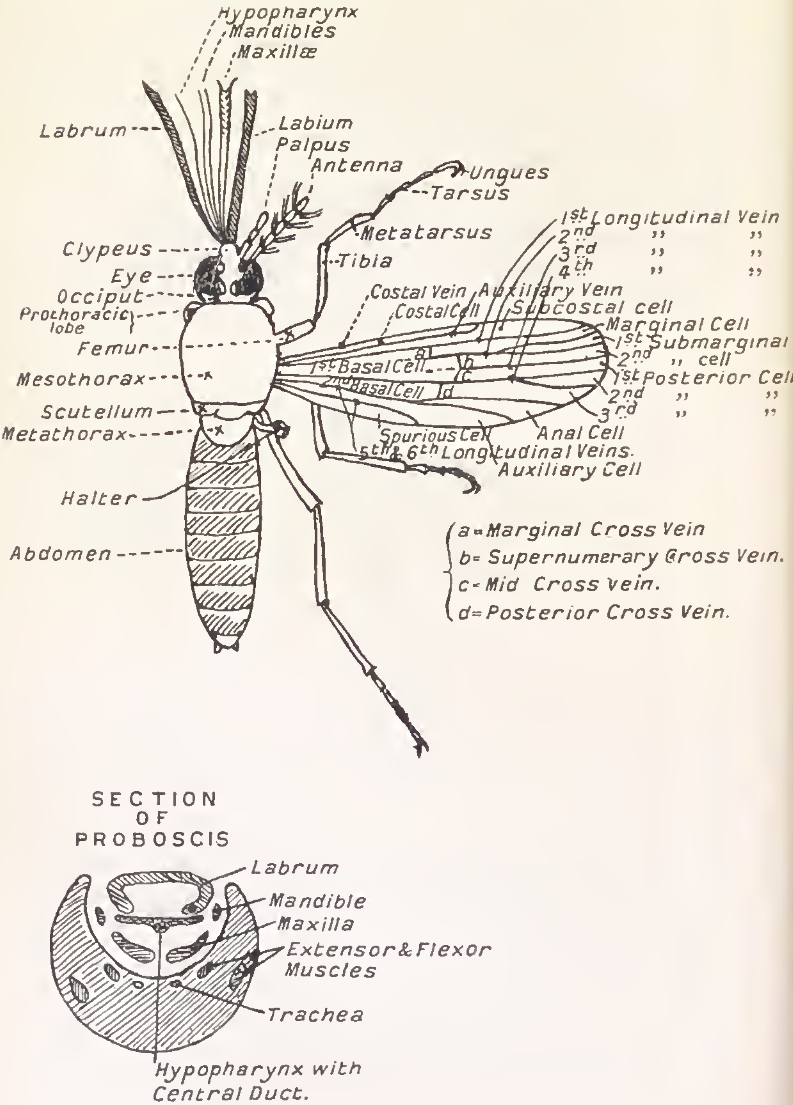

Fig. 20.—Anatomy of the Mosquito.

Anatomy of the Adult Mosquito.

I. **The Head.**—The following parts can be made out (Fig. 20):—

(*a*) Two large compound *eyes*.
(*b*) The *occiput*, between and above the eyes.
(*c*) The *vertex*, between and in front of the eyes or frons.
(*d*) The *genæ*, or sides of the head.
(*e*) The *clypeus*, a blunt anterior projection. This may be simple, ridged, nude, hairy, or scaly.
(*f*) The *Proboscis*:—

Labrum or upper lip.

Hypopharynx, a single-pointed cannula lying in the groove of the labrum. It is pierced by the salivary duct.

Two maxillæ, lancet needles with spiked barbs, on each side of hypopharynx.

Two mandibles, plain lancet needles lying by the maxillæ.

Labium, or lower lip, a sheath enclosing the other parts. It is fleshy and scale-covered.

(*g*) *Two palpæ*, or sensory organs, with two or more segments. They vary greatly in size, shape, and length.
(*h*) *Two antennæ*. They have a basal segment and a polyarthritic flagellum which is usually plumose in the male and covered with short hairs in the female.

II. **The Thorax** is composed of three segments rigidly fused together. The mesothorax occupies the largest part, and the tergum of the mesothorax is almost the only visible part when the insect is viewed from above. From each of the three divisions spring a pair of legs. All parts of the thorax may be scale-covered, but the metathorax is usually nude. The following parts should be identified:—

(*a*) The *prothorax*, consisting of two lateral prothoracic lobes.
(*b*) The *mesothorax* or *mesonotum*, with a sharply restricted piece at the posterior, called
(*c*) The *scutellum*, which may be rounded or tri-lobed.
(*d*) The *metathorax* or *metanotum*, a rounded extremity of the thorax, situated under the scutellum.
(*e*) The *pleura* or "breast side" of the thorax.
(*f*) The *propleura* or front part of the pleura, from which springs the front leg.
(*g*) The *mesopleura* or central portion of the pleura. This carries the stigma, the middle leg, and the wings.
(*h*) The *metapleura* or posterior portion of the pleura, from which springs the coxa of the hind leg. Just above, and attached to, the base of the metathorax are—

(*i*) The *halteres* or poisers, representing rudimentary hind wings. They are a pair of small club-like organs, consisting of a stem and a pear-shaped head.

(*j*) The *legs*, each consisting of nine segments. Of these the basal attachment to the body is called the *coxa;* followed by the *trochanter*, *femur*, *tibia*, *metatarsus*, and four jointed *tarsus* ending in two claws or *ungues.*

(*k*) The *wings*, which have six *longitudinal veins* (except in the *Heptaphlebomyinæ*, which have seven). The *costal vein* runs round the whole border of the wing. For the names of veins and cells, see Fig. 20.

III. **The Abdomen** has eight segments. In the female it terminates in two lobes; in the male by claspers, &c. The abdomen is often scaly. There is a border of bristles along the posterior edge of each segment.

Fig. 21.—Culicine Scales.

1 to 3, Head scales. 4 to 8, Thoracic scales.
9 to 13, Wing-vein scales.

IV **The Scales.**—The grouping of generic characters is determined by the scale type, which is, therefore, of importance.

(a) *Head scales* (1 to 3, Fig. 21):—

i. Narrow, curved.
ii. Upright, forked.
iii. Flat or spatulate.
iv. Spindle-shaped } Rare.
v. Twisted }

(b) *Thoracic scales* (4 to 8, Fig. 21):—

i. Narrow, curved.
ii. Hair-like, curved.
iii. Spindle-shaped.
iv. Flat, spatulate.
v. Twisted.

(c) *Abdominal scales*:—

i. Flat, spatulate (in most cases).
ii. Spindle-shaped (*Cellia*).
iii. Narrow, curved (*Pyretophorus*).
iv. Twisted and upright (*Mucidus*).

(d) *Wing scales* (9 to 13, Fig. 21):—

i. Narrow, straight, and linear *Culex*).
ii. Short, broad, and spatulate (*Melanoconion*).
iii. Broad and straight (*Tæniorhynchus*).
iv. Broad and asymmetrical (*Mansonia*).
v. Heart-shaped (*Etorleptiomyia*).
vi. Lanceolate (*Anopheles* and *Pyretophorus*).
vii. &c.

Examples of some of these scales will be seen in Fig. 21.

Internal Anatomy (Fig. 22).—The food juices are sucked p the tube formed by the labrum and hypopharynx. When the iosquito bites, some saliva is injected into the wound by a small uct which runs in the hypopharynx.

The various mouth parts, mentioned previously, coalesce behind ie clypeus.

Here there is a *buccal cavity* opening into the pharynx by valvular arrangement.

The *pharynx* extends from the mouth to the œsophagus, and is ie pumping organ by which the mosquito sucks up juices.

The *œsophagus* is a short tube commencing at the end of the harynx, at about the level of the neck, and extending to the -sophageal valve which connects with the mid-gut. Opening ito this œsophagus are three large blind sacs which *Nuttall* and *hipley* have shown to be food reservoirs. One is ventral, and ktends back, when full, to the seventh segment. The other two re latero-dorsal.

The *œsophageal valve*, answering to the proventriculus of other isects, is a kind of valvular tube containing a variable number of xcrescences, and serves to connect the œsophagus with the mid-gut.

The *mid-gut* is a straight tube running from the œsophageal valve to its junction with the hind-gut at about the level of the sixth segment. The posterior dilated portion of the mid-gut (in which the malarial parasites develop) is called the *stomach*.

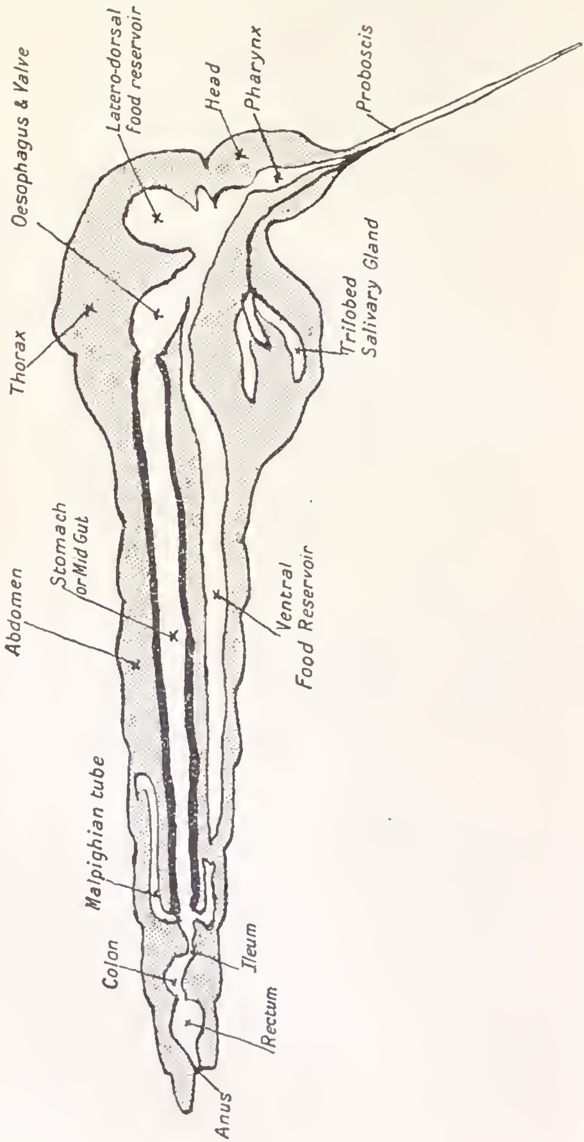

Fig. 22.

The *hind-gut* is short, and extends to the anus. It is arbitrarily ivided into three portions—ileum, colon, and rectum. Into the ind-gut, at its junction with the stomach, open five long, con-oluted, darkish glandular bodies—the *Malphigian tubes*—which re supposed to have an excretory function.

The *salivary glands*, by which malarial, filarial, yellow fever, and ossibly other infections (such as dengue) are transmitted to man, onsist of two tri-lobed structures situated on each side of the norax. From each of these structures a duct passes forward to the evel of the buccal cavity, where the two ducts anastomose to form common duct. This common salivary duct is connected with the alivary groove in the hypopharynx by a chitinous receptacle, acting s a pump, and actuated by powerful voluntary muscles.

The salivary duct does not communicate with the alimentary anal.

Each of the tri-lobed glands consist of three cæcal tubules, two of hich secrete the saliva, and the third, an irritating fluid, supposed o prevent the coagulation of the abstracted blood.

The *respiratory system* is of the usual type. There are no igmata in the head or last abdominal segment. The two anterior oracic ones are the largest and most important. The openings re small and much hidden by scales.

The main trunks from the stigmata give off communicating ranches to those before and behind, and end in a tuft of branches or the supply of muscles and organs.

The genital system, in the male, consists of a pair of testes pening by vasa deferentia into the ejaculatory duct. The enis is situated between the two claspers, and is a soft and eshy organ.

In the female, there are two ovaries connected with a common uct by two Fallopian tubes.

Spermathecæ, for storing the spermatozoa, are connected with is common duct by a tubule of small calibre.

Dissection of Mosquito's Stomach.—Deprive the mosquito of s wings and legs, and place it on a slide in saline solution. Make small nick with the point of a needle on each side of the last but ne abdominal segment.

Fix the point of one needle on the thorax; lay the shaft of second needle on the last abdominal segment, and exert gentle action. The weakened exoskeleton gives way at the point reviously notched, and the intestine can be then gently drawn out or some distance. Then decapitate the insect, when the rest of e alimentary canal can be easily withdrawn, and removed for xamination.

To *dissect out the salivary* glands needs care.

The back of the thorax is separated by longitudinal incision with cataract knife. At the level of the second pair of legs an incision made at right angles to the previous one. The remnant of the orax is now fixed with a needle, while gentle traction is made on

the neck with a second needle. The salivary glands are thus pulled out of their bed, and will be found attached to the head.

A second method is to cut across the thorax at the level of the second pair of legs, and decapitate the insect. Leave the anterior portion of the thorax, thus isolated, in the saline solution, and mount. A little practice is necessary to perform the manœuvre with constant success, and pressure may have to be exerted on the cover-glass to squeeze out the glands from underneath portions of the exoskeleton.

Classification (after Theobald).

SUB-FAMILIES OF

CULICIDÆ.

- 1. **Anophelinæ.** Proboscis straight; Palpi long in both sexes.
- 2. *Orthorhynchæ.* Proboscis straight; Palpi short in ♀, variable in ♂.
  - *Metanotopsilæ.* (Metanotum nude.)
    - *Heteropalpæ.* Palpi long in ♂.
      - **Culicinæ.** Wings with six long veins.
      - **Heptaphlebomyinæ.** Wings with even long veins.
    - *Micropalpæ.* Palpi short in both.
      - **Œdinæ.** Abdomen simple.
      - **Hæmagoginæ.** Abdomen notched below.
  - *Metanototrichæ.* (Metanotum with scales or chætæ.)
    - *Heteropalpæ.* Palpi long in ♂.
      - **Hylo-conopinæ.**
    - *Micropalpæ.* Palpi short in both.
      - **Dendro-myinæ.**
- 3. *Ankylorhynchæ.* Proboscis curved.
  - **Toxorhynchinæ.** Palpi short in ♀.
  - **Megarhininæ.** Palpi long in both sexes.

TABLE OF GENERA.

SUB-FAMILY Anophelinæ (Theobald).

Genus 1. **Anopheles*** (Meigen).
2. **Myzomyia*** (Blanchard).
3. *Cycloleppteron* (Theobald).

4. *Stethomyia* (Theobald).
5. **Pyretophorus*** (Blanchard).
6. *Arribalzagia* (Theobald).
7. **Myzorhynchus*** (Blanchard).
8. *Christya* (Theobald).
9. *Lophoscelomyia* (Theobald).
10. **Nyssorhynchus*** (Blanchard).
11. **Cellia*** (Theobald).
12. *Aldrichia* (Theobald).
13. *Kertészia* (Theobald).
14. *Bironella* (Theobald).
15. *Neocellia* (Theobald).
16. *Myzorhynchella* (Theobald).

SUB-FAMILY **Ankylorhynchæ** (Lutz).

Toxorhynchinæ (Theobald).

Genus 1. *Toxorhynchites* (Theobald).

Megarhininæ (Theobald).

Genus 2. *Megarhinus* (Fabricius).
3. *Ankylorhynchus* (Lutz).

SUB-FAMILY **Orthorhynchæ** (Lutz).

METANOTOPSILÆ (Lutz).

(*a*) **Culicinæ** (Theobald).

Genus 1. *Theobaldia* (Blanchard).
2. *Lutzia* (Theobald).
3. *Mucidus* (Theobald).
4. *Mansonia* (Blanchard).
5. *Tæniorhynchus* (Arribalzaga).
6. *Melanoconion* (Theobald).
7. *Grabhamia* (Theobald).
8. *Pseudograbhamia* (Theobald).
9. *Acartomyia* (Theobald).
10. *Psorophora* (Desvoidy).
11. *Janthinosoma* (Arribalzaga).
12. *Desvoidea* (Blanchard).
13. *Eretmapodites* (Theobald).
14. **Stegomyia*** (Theobald).
15. *Pseudoskusea* (Theobald).
16. *Scutomyia* (Theobald).
17. *Œdimorphus* (Theobald).
18. *Leicesteria* (Theobald).
19. *Macleaya* (Theobald).
20. *Hulecotomyia* (Theobald).

21. *Phagomyia* (Theobald).
22. *Polyleptiomyia* (Theobald).
23. *Howardina* (Theobald).
24. *Danielsia* (Theobald).
25. *Lepidotomyia* (Theobald).
26. *Catageiomyia* (Theobald).
27. *Finlaya* (Theobald).
28. *Trichorhynchus* (Theobald).
29. *Quasi-stegomyia* (Theobald).
30. *Bancroftia* (Lutz).
31. **Culex*** (Linnæus).
32. *Lophoceratomyia* (Theobald).
33. *Trichopronomyia* (Theobald).
34. *Lasioconops* (Theobald).
35. *Gilesia* (Theobald).

(*b*) **Heptaphlebomyinæ** (Theobald).

36. *Heptaphlebomyia* (Theobald).

(*c*) **Aedinæ** (Theobald).

37. *Aedes* (Meigen).
38. *Aedinus* (Lutz).
39. *Aedeomyia* (Theobald).
40. *Deinocerites* (Theobald).
41. *Mimomyia* (Theobald).
42. *Uranotænia* (Arribalzaga).
43. *Anisocheleomyia* (Theobald).
44. *Ficalbia* (Theobald).
45. *Leptosomatomyia* (Theobald).
46. *Polylepidomyia* (Theobald).
47. *Verrallina* (Theobald).
48. *Rhachionotomyia* (Theobald).
49. *Etorleptiomyia* (Theobald).

(*d*) **Hæmagoginæ** (Lutz).

50. *Hæmagogus* (Williston).
51. *Gualteria* (Lutz).

B. METANOTOTRICHÆ (Lutz.)

(*a*) **Hyloconopinæ** (Lutz).

52. *Trichoprosopon* (Theobald).
53. *Joblotia* (Blanchard).
54. *Gœldia* (Theobald).
55. *Runchomyia* (Theobald).
56. *Hyloconops* (Lutz).

(*b*) **Dendromyinæ** (Lutz).

57. *Dendromyia* (Theobald).
58. *Wyeomyia* (Theobald).
59. *Phoniomyia* (Theobald).
60. *Sabethes* (Desvoidy).
61. *Sabethoides* (Theobald).
62. *Sabethinus* (Lutz).
63. *Limatus* (Theobald).

* Only those *genera* in heavier type and starred in the above list are at present known to be of medical interest.

For more detailed study it is advisable to consult Theobald's *Monograph of the Culicidæ of the World.*

The following brief abstract from Theobald may prove of use in looking up points connected with the chief genera.

Anophelinæ.—This sub-family has long palpi in both sexes; that thoracic and scutellar scales are absent; the proboscis is straight; the wings (except those of *Anophelis bifurcatus* and *Stethomyia nimba*) are spotted; and the resting position (except that of *Myzomyia culicifacies*) is almost at right angles to the surface of support.

The metanotum is always nude, and the scutellum is always simple—not tri-lobed as in other *Culicidæ*.

The larvæ have no respiratory siphon, and lie parallel with the surface of the water.

There are 115 species of this sub-family which are at present known. Some are sylvan; others, domesticated.

The various genera of the Anophelinæ may be thus differentiated:—

(*a*) Thorax and abdomen with hair-like curved scales.

(*α*) Head with upright forked scales only.

1. Wing-scales large and lanceolate, wing unspotted or collections of similar coloured scales.

   Genus:—*Anopheles.*

2. Wing-scales lanceolate, but small. Spotting of varied colour.

   Genus:—*Myzomyia.*

3. Wings with patches of large inflated scales.

   Genus:—*Cycloleppteron.*

(*β*) Median area of head with flat scales.

Genus:—*Stethomyia.*

(*b*) Thorax with narrow, curved scales. Abdomen hairy. Wing-scales small and lanceolate.

Genus:—*Pyretophorus.*

(*c*) Thorax with hair-like curved scales. Abdomen with lateral scale tufts, and scaly venter. Wings-scales lanceolate.

Genus:—*Arribalzagia*.

(*d*) Thorax with hair-like curved scales. No lateral abdominal tufts. Apical ventral tuft, and dense-scaled palpi in the ♀. Wings with dense, large, lanceolate scales.

Genus:—*Myzorhynchus*.

(*e*) Thorax with hair-like curved scales, and narrow curved lateral ones. Abdomen hairy, with lateral apical scaly tufts. Wing-scales short, lanceolate, and dense. Fork-cells short.

Genus:—*Christya*.

(*f*) Thorax with very long hair-like curved scales. Abdomen hairy; last two segments scaly. Hind femora densely scaly. Wings broad, blunt, lanceolate scales.

Genus:—*Lophoscelomyia*.

(*g*) Thorax and abdomen with scales.

(α) Thoracic, spindle-shaped; abdominal, as lateral tufts, and small dorsal patches of flat scales.

Genus:—*Nyssorhynchus*.

(β) Abdomen almost completely scaled with long irregular scales and lateral scale tufts.

Genus:—*Cellia*.

(γ) Abdomen completely scaled with large flat scales as in *Culex*.

Genus:—*Aldrichia*.

SYSTEMATIC NOTES ON GENERA.

A. ANOPHELINÆ.

Genus **Anopheles**.

Habitat.—Europe, N. Africa, Hills of India, Australia, W. Indies, N. America, and W. Africa.

Type species.—*A. maculipennis* (Europe and N. America).

No. of known species 15.

Spotted wing species.—Scanty in several species, such as *A. crucians* (Wiedemann), *A. gigas* (Giles).

Malaria carriers.—*A. maculipennis*, *A. algeriensis* (and possibly all the other species).

Larvæ.—*A. maculipennis* may be found in rain-water barrels, &c.

Hibernation.—A. maculipennis can hibernate as adult, *A. bifurcatus* can hibernate as larva.

Genus **Myzomyia**.—Small mosquitoes with spotted wings and aried habits.

Type species.—M. funesta.

Malaria carriers.—M. listonii.
M. funesta.
M. turkhudii.
M. culicifacies.
M. nili.

River breeders.—M. listonii.
M. nili.
M. funesta.

Habits.—M. culicifacies adopts the culicine position when resting.

Species 1. *M. funesta* (Giles).—C. and W. Africa, Sudan, Philippines.
2. *M. rossii* (Giles).—India, F.M.S., Philippines.
3. *M. ludlowii* (Theobald).—Philippines, Malaya.
4. *M. rhodesiensis* (Theobald).—C. Africa.
5. *M. culicifacies* (Giles).—India.
6. *M. listonii* (Liston).—India, F.M.S.
7. *M. longipalpis* (Theobald).—C. Africa.
8. *M. leptomeres* (Theobald).—India.
9. *M. lutzii* (Theobald).—Brazil. Brit. Guiana.
10. *M. turkhudii* (Liston).—India.
11. *M. hispaniola* (Theobald).—Spain, Madeira.
12. *M. elegans* (James and Theobald).—India.
13. *M. punctulata* (Dönitz).—Sumatra, Borneo, N. Guinea.
14 *M. tessellata* (Theobald).—Str. Settlements.
15. *M. leucosphyra* (Dönitz).—Sumatra, Borneo, N. Guinea.
16. *M. albirostris* (Theobald).—F.M.S.
17. *M. nili* (Theobald)—Sudan.
18. *M thorntonii* (Ludlow).—Philippines.
19. *M. aconita* (Dönitz).—Sumatra, Java.
20. *M. hebes* (Dönitz).—E. Africa.
21. *M. pyretophoroides* (Theobald).

Genus **Cyclolepppteron**.

Species 1. *C. grabhamia* (Theobald).—Jamaica.
2. *C. mediopunctata* (Theobald).—Brazil.

Genus **Stethomyia**.

Species 1. *S. nimba* (Theobald).—S. America.
2. *S. fragilis* (Theobald).—F.M.S.

Genus **Pyretophorus**.—Large mosquitoes, with spotted wings found only in Africa, Asia, and Australia.

Breeding habits.—Larvæ found in flowing water as well as in ordinary situations.

Malaria carriers.—P. costalis.
P. chandoyei.
P. ardensis.

Type.—P. costalis.

Species 1. *P. costalis* (Loew).—Africa, Mauritius.
2. *P. marshalli* (Theobald).—Mashonaland.
3. *P. minimus* (Theobald).—Hongkong.
4. *P. ardensis* (Theobald).—Natal.
5. *P. chandoyei* (Theobald).—Algeria.
6. *P. superpictus* (Grassi).—S. Europe.
7. *P. palestinensis* (Theobald).—Palestine, Cyprus.
8. *P. jeypurensis* (Theobald).—India.
9. *P. cinereus* (Theobald).—S., W., and C. Africa.
10. *P. atratipes* (Skuse).—N. S. Wales, Queensland.
11. *P. lutzii* (Cruz).—Brazil.

Genus **Arribalzagia**.

Species 1. *A. maculipes* (Theobald).—Brazil, Trinidad.

Genus **Myzorhynchus**.—Large, dark mosquitoes, generally with white on the legs. Palpi and proboscis densely scaled. They are wild insects, frequenting especially overgrown dark pools. They bite viciously, but their connection with malaria has not been proved. The parasites can, however, develop in them; and *M. nigerrimus* has been proved an efficient host for *Filaria bancrofti.*

Species 1. *M. barbirostris* (Van der Wulp).—India, W. Africa.
2. *M. bancrofti* (Giles).—Queensland.
3. *M. umbrosus* (Theobald).—Malaya.
4. *M. albotæniatus* (Theobald).—Str. Settlements.
5. *M. Sinensis* (Wiedemann).—China, Formosa, F.M.S.
6. *M. vanus* (Walker).—Malaya, China.
7. *M. annularis* (Van der Wulp).—Java, Malaya, India.
8. *M. pseudopictus* (Grassi).—Italy.
9. *M. minutus* (Theobald).—India.
10. *M. nigerrimus* (Giles).—India.
11. *M. mauritianus* (Grandpré and Charmoy).—Mauritius, C. and N. Africa.
12. *M. plumiger* (Dönitz).—Hongkong, India.
13. *M. paludis* (Theobald).—W., C., and N. Africa.
14. *M. pseudobarbirostris* (Ludlow).—Philippines.
15. *M. coustanii* (Laveran).—Madagascar.
16. *M. strachanii* (Theobald).

Genus **Christya** (Theobald).—These are *very long* apical lateral tufts of scales on the abdomen.

Species 1. *C. implexa* (Theobald).—Uganda.

Genus **Lophoscelomyia** (Theobald).—Only known as yet in Malaya. The larvæ live in bamboo hollows.

Species 1. *L. asiatica* (Leicester).—Malaya.

Genus **Nyssorhynchus.**—They are of both jungle and domestic habits, and are both pot, puddle, and marsh breeders.

The connection with malaria is not proved, but parasites will develop in some of the species.

Species 1. *N. maculatus* (Theobald).—India, Malaya.
2. *N. theobaldii* (Giles).—India, Aden.
3. *N. stephensii* (Liston).—India.
4. *N. fuliginosus* (Giles).—India, F.M.S.
5. *N. maculipalpis* (Giles).—India, Mauritius, Mashonaland.
6. *N. pretoriensis* (Theobald).—Pretoria, Natal.
7. *N. witmorii* (James).—Kashmir, Malaya.
8. *N. karwarii* (James).—Karwar, Goa, Malaya.
9. *N. annulipes* (Walker).—Australia.
10. *N. masterii* (Skuse).—Australia.
11. *N. nivipes* (Theobald).—F.M.S.
12. *N. jamesii* (Theobald).—S. India, Ceylon.
13. *N. philippinensis* (Ludlow).—Philippines.

Genus **Cellia** (Theobald).—Some of these are bush mosquitoes breeding in pools or any open water. Others, as *C. pharoensis*, *C. pulcherrima*, and *C. argyrotarsus*, may be found in houses, but, unless enclosed during the night, they may not be found at daybreak.

Malaria carriers.—*C. pharoensis.*
C. argyrotarsus.

Species 1. *C. pharoensis* (Theobald).—African Continent.
2. *C. pulcherrima* (Theobald).—India.
3. *C. squamosa* (Theobald).—African Continent.
4. *C. kochii* (Dönitz).—Malay Peninsula and Archipelago, Philippines.
5. *C. argyrotarsus* (Robineau-Desvoidy).—W. Indies, C. and S. America.
6. *C. albimanus* (Wiedemann).—W. Indies, Br. Guiana, Brazil.
7. *C. bigotii* (Theobald).

Genus **Aldrichia** (Theobald).—The abdomen is completely covered with flat overlapping scales, as in Culex.

Species 1. *A. error* (Theobald).—India.

Genus **Bironella** (Theobald) differs from all the preceding *Anophelinæ* in having the fork cells very small, instead of long.

Species 1. *B. gracilis* (Theobald).—New Guinea.

B. CULICINÆ.

Features.—Straight probosces. Palpi short in females. Metanotum nude. Six longitudinal wing veins. Larvæ all have siphons.

Genus **Theobaldia.**—Species of this genus often have spotted wings, and may be confounded with *Anophelinæ*. They are usually of domestic habit and temperate distribution. *T. annulata* and *T. spathipalpis* are frequently found in privies. The adults hibernate.

Species 1. *T. annulata* (Schrank). — Europe, India, N. America.
2. *T. ficalbii* (Noé).—Italy.
3. *T. glaphyropetra* (Schiner).—Austria.
4. *T. incidens* (Thomson).—California, New Mexico.
5. *T. spathipalpis* (Rondani).—Mediterranean Littoral, N. Africa, India.
5. *T. penetrans* (Desvoidy).—France.

Genus **Mucidus** (Theobald).—The species have a mouldy appearance, due to twisted grey scales. The larvæ breed in both salt and fresh water, and are carnivorous.

Species 1. *M. africanus* (Theobald).—Africa.
2. *M. mucidus* (Karsch).—Africa.
3. *M. alternans* (Westwood).—Australia.
4. *M. laniger* (Wiedemann).—India.
5. *M. scatophagoides* (Theobald).—India.

Genus **Mansonia** (Blanchard).—They have broad, flat, mottled, asymmetrical wing scales. The ova have attenuated necks and are laid separately. The insect is an exceptional pest along the Nile.

Species 1. *M. titillans* (Walker). — S. and C. America, W. Indies.
2. *M. pseudotitillans* (Theobald).—S. and C. America, W. Indies.
3. *M. amazonensis* (Theobald).—S. and C. America, W. Indies.
4. *M. uniformis* (Theobald).—Africa, Asia.
5. *M. major* (Theobald).—Africa.
6. *M. nigra* (Theobald).—Africa.
7. *M. annulifera* (Theobald).—Asia.
8. *M. annulipes* (Walker).—Asia.

Genus **Melanoconion** (Theobald).—These are very small black mosquitoes which can pierce an ordinary mosquito net They are both domestic and sylvan. The larvæ occur in permanent pools, feed on algæ, and are often of green colour.

Species 1. *M. atratus* (Theobald).—W. Indies, S. America.
2. *M. spisipes* (Theobald).—W. Indies.
3. *M. nigripalpus* (Theobald).—W. Indies.

Genus **Grabhamia**.—The wing-scales are parti-coloured, giving the wings a pepper-and-salt appearance.

The eggs are often laid on damp mud. The larvæ have short siphons, but lie nearly parallel with the water surface.

About fifteen species are known. They are mostly from N. America and Europe; one is from Natal and one from the Philippines.

G. dorsalis (Meigen) is most common in Europe, and is found in the Thames valley and along the E. coast of Britain. One species (*G. idahœnsis*) is sufficiently small to get through a mosquito net.

Genus **Stegomyia** (Theobald).—This is a highly important group. A name frequently given to them is that of "Tiger Mosquito." They are small and dark, with silver markings and banded legs. The scutellum has always dense flat scales.

Most of the species can be identified by the thoracic markings.

There are both sylvan and domestic species, and most of them are both nocturnal and diurnal in habit.

The ova are oval and black, are deposited singly, and can withstand long desiccation.

Eighteen species are known, and their distribution is very wide. In most parts of the world between the latitudes of 40° N. and 40° S. representatives may be found.

S. fasciata (Fabricius), the old *Culex fasciatus*, is the carrier of yellow fever. It may be recognised by the lyre-shaped adornment of the thorax—two curved silvery lines, separated by two median yellow lines. It breeds in any domestic water supply. It usually rests on any dark material or in dark corners during the day, but will often bite by day as well as by night.

It is quite possible that others of this genus may be able to transmit yellow fever, though this has not yet been worked out.

There is one species widely distributed in Asia and Oceania—*S. scutellaris* (Walker). This species much resembles *S. fasciata*, but has a single median silvery line on the thorax instead of the lyre-shaped adornment.

Species 1. *S. fasciata* (Fabricius).—Between 40° N. and S. of Equator.
2. *S. scutellaris* (Walker).—Asia, Oceania.
3. *S. africana* (Theobald).—W. and C. Africa.
4. *S. thomsonii* (Theobald).—India.
5. *S. grantii* (Theobald).—Socotra.

6. *S. nigeria* (Theobald).—W. Africa.
7. *S. crassipes* (Van der Wulp).—Burmah.
8. *S. argenteopunctata* (Theobald).—Mashonaland.
9. *S. punctolateralis* (Theobald).—Queensland.
10. *S. signifer* (Coquillett).—N. America.
11. *S. amesii* (Ludlow).—Philippines.
12. *S. W-alba* (Theobald).—India.
13. *S. pseudonivea* (Theobald).—Singapore.
14. *S. simpsonii* (Theobald).—Transvaal.
15. *S. powerii* (Theobald).—Natal.
16. *S. annulisostris* (Theobald).—Ceylon.
17. *S. mediopunctata* (Theobald).—Ceylon.
18. *S. brevipalpis* (Giles).—India.

Genus **Culex.**—This has a very world-wide distribution. The characters, as given by Theobald, are :—Head with narrow, curved scales above, flat at sides, and with upright forked scales.

Male palpi long and acuminate No flat thoracic scales, except on the pleuræ.

Fork cells long, lateral vein scales linear. Male genitalia have a lateral leaf-like process.

At present there are about 250 species placed in this genus, which will undergo subsequent modification. The following species is the chief one of medical importance :—

C. fatigans (Wiedemann).—This is the tropical brown house mosquito, and is the main intermediate host of *Filaria bancrofti*, &c. It is said to be possibly the carrier also of dengue, but several cases noted by the author would seem rather to point to *Stegomyia*.

Its distribution is very wide and of much the same limits as those of *Stegomyia fasciata*.

The thoracic scales are golden brown, with two dark median lines on the mesonotum. The abdomen also has brown scales, with basal white lateral spots, and creamy basal bands to all segments. The legs are uniformly brown. The wings have brown linear lateral scales, and the first fork cell is always longer than the second.

The eggs are laid in rafts. The larvæ have a fairly long siphon, and feed on algæ.

Other sub-families and their genera are not of sufficient medical importance to be dealt with in this volume.

CHAPTER X.

NOTES ON FLEAS AND TICKS.

FLEAS.

THE connection of fleas with the transmission to man of bubonic plague, and possibly of other diseases, renders the study of them most important to the medical biologist or epidemiologist.

There are two books in which to study the subject:—1. *Die Flöhe* by Taschenberg. 2. American Siphonaptera (*Proc. U.S. Nat. Mus.*, vol. xxviii.), by C. F. Baker.

Fleas for identification should be sent to Hon. N. C. Rothschild, Tring Park, Herts.

Anatomy of the Flea.—The flea is almost peculiar in being laterally compressed.

The *head* is of peculiar shape, small, and not distinctly separated from the body. The *clypeus* is absent.

The *antennæ* are placed in fossæ behind the eyes, and consist of two basal joints, loosely connected with which is a terminal segmented mass of irregular shape.

The *mouth* is differently constructed to that of any other insect.

It consist of:—(*a*) A central stylet—the *hypopharynx*—serrated above and tubular below.

(*b*) Two serrated *mandibles*, excavated on the inner side, and forming with (*a*) a hollow tube.

(*c*) A single *labium* which bifurcates to form two labial palps, which serve as a shield for (*a*) and (*b*).

(*d*) Two flattened plate-like *maxillæ*, each having a four-jointed palpar extremity.

The *thorax* consists of three separate segments.

The *metanotum* has a flap or epiphysis, thought to be a homologue of wings.

The full number of ten *stigmata* exists—three thoracic and seven abdominal.

The *abdomen* has ten overlapping segments, and the lack of continuity between the dorsal and ventral integument permits of considerable distension. The hinder segments are differentiated to form the genital apparatus.

The *front legs* have a large additional basal piece (sometimes called the Ischium), which at first sight gives the impression that they spring from the head.

Life History and Habits.—The female lays about a dozen *eggs* in the hair or fur of an animal host, or in cracks of the floor, &c. In many species these ova are about 0·5 mm. in length.

In about a week or more they hatch out into *larvæ*. The young larva bears on the head a curious structure for breaking the egg-shell. They are whitish, worm-like bodies of fourteen segments. They have the mouth parts of a mandibulate insect, and they are peripneustic—having ten pairs of stigmata.

They feed on any organic refuse (or blood); and in about eleven days are full grown, when the larva makes a cocoon. Seven to fourteen days thereafter, the perfect *flea* emerges. Thus the whole evolution takes from four to six weeks.

There are over a hundred species of fleas, all of which are parasitic on mammals or birds.

It has been frequently said that each species of mammal has its peculiar flea. As a general statement this is not far wrong, but it is by no means the case in every instance. For example, out of 100 fleas found on human beings, about 97 will prove to be *Pulex irritans*. On the black rat (*Mus rattus*) the majority of fleas found will be *P. cheopis*, though not in such large percentage as mentioned for *P. irritans* above. With equal prevalence *P. fasciatus* will be found on the large brown rat (*Mus decumanus*).

There is, however, a flea (*P. serraticeps* or *P. felis*) which exhibits no invidious distinctions in the choice of a host. This flea is the commonest of all fleas, and may be found at almost any time on the following amongst other hosts:—Cats, dogs, rats, monkeys, sheep, deer, goats, horses, &c. But, even although found on such manifold hosts, this flea does not, as a rule, go from one to the other, since it objects to being dislodged from comfortable quarters.

When a host dies, or often when it is only ill, the fleas leave it in a body. On the cold corpse of an animal, it is extremly rare to find a single flea left, even though, during life, the animal had been infested with them. Such fleas that have departed seek a similar host in the neighbourhood. If, as in the case of plague-stricken rats, all other similar hosts have left the neighbourhood, then the flea will seek the nearest warm-blooded host available. Thus, if a person enter a closed and empty room which had previously been occupied by cats or dogs, it may often happen that he is viciously attacked by fleas, which, if examined, will generally prove to be the cat and dog flea—*P. serraticeps*.

This subject is of great interest in connection with the spread of plague (*vide* Chapter xxx.).

Most fleas have a nocturnal habit of feeding. *P. serraticeps* is the least so, and will feed at any time as witnessed by the tortures of our domestic pets.

Classification.—The following brief scheme will show some of the more important genera:—

A. Thoracic segments short and narrow. Labial palpi without pseudo-joints. Third antennal segment without pseudo-joints.

(*a*) Maxillæ with absent, or very short, projecting laminæ. Maxillary pulps extending beyond anterior coxæ. Head produced into sharp point in both sexes. Thoracic flaps extending over two or three abdominal segments.

α. Maxilla without projecting lamina. Angle of head produced. Thoracic flaps over three abdominal segments. No heavy spines to fifth tarsal segment. Legs almost spineless.

Genus:—*Sarcopsylla.*

β. Maxilla with short projecting lamina. Angle of head not produced. Thoracic flaps over two abdominal segments. Fifth tarsal joint with usual spines. Legs spinous.

Genus:—*Xestopsylla.*

(*b*) Maxillæ with long, narrow, curved lamina. Maxillary flaps as long as anterior coxæ. Head evenly round. Thoracic flaps over one abdominal segment.

Genus:—*Hectopsylla.*

B. Thoracic segments not short and narrow. Labial palps with three or more pseudo-joints. Third antennal segment with nine or more pseudo-joints. Maxillary palps shorter than anterior coxa. Thoracic flaps over only one abdominal segment.

(*a*) Labial palps with 11 to 13 pseudo-joints. Gravid abdomen much swollen. Antepygidial bristles absent.

Genus:—*Vermipsylla.*

(*b*) Labial palps with three to five pseudo-joints. Antepygidial bristles present.

α. Black teeth, posterior of tibia i. Fifth tarsal segment (i) greatly enlarged. Claws as long as fifth joint. Only few long spines on coxæ i. Gravid abdomen swollen.

Genus:—*Megapsylla.*

β. Slender spines, posterior of tibia i. Fifth tarsal segment (i) not greatly enlarged. Numerous rows of bristles on coxæ i. Gravid abdomen does not expose interscleritic membrane. Max-

illæ long triangular: apex acute. No ctenidia to abdominal segments. Posterior tibial spines in pairs. Row of four stout spines on last segment on all tarsi. Eyes large. One antepygidial bristle on each side in female.

(1) Head without ctenidia.

Genus:—*Pulex*.

(2) Head with ctenidia.

Genus:—*Ctenocephalus*.

TICKS.

That ticks have been proved to act as efficient hosts for the organism of the "Spirillar-fever" of human beings, as well as for the Texas-fever of cattle, has invested them with an importance previously undreamed of.

Ticks and mites, which make up the order *Acari*, are the only parasites of the class *Arachnida*.

The Order Acari is divided into two families.

i. *Argasidæ*.
ii. *Ixodidæ*.

The majority of ticks which are harmful to man belong to the first of these two families, and of these many species are parasitic on domestic fowls.

Ticks may be readily distinguished from insects, because they have—

1. No antennæ.
2. Four pairs of legs.
3. No differentiation between thorax and abdomen.

Life History.—The fertilised female leaves her host, drops to the ground, seeks some secluded spot, and there lays her eggs.

After laying several thousand eggs during the course of a week or two, the female dies.

In some two months or less the eggs hatch into six-legged larvæ with no spiracular or genital orifices. These larvæ climb bushes or shrubs with the hope of attaching themselves to some passing warm-blooded host. This object being attained, they feed for a few days before dropping to earth, where is passed a resting stage of several weeks preparatory to moulting. At the latter operation a *nymph* emerges—an eight-legged individual, with spiracular, but no genital, orifices.

This pupa repeats the wiles of the larva, and, after feeding on the blood of the new host for a week or so, it drops to the ground and hides for a few weeks till the adult is produced. This mature tick

has both genital and spiracular orifices, and is furnished with four pairs of legs.

The quest for a host is a repetition of the procedure adopted by the larva and pupa.

Conjunction of the sexes occurs after a short feed, and is accomplished on the part of the male by the insertion of his mouth parts into the genital orifice of the female, which lies at the middle or anterior part of the ventral surface.

The female then leaves her host to seek a place to deposit her eggs; the males, however, may remain on the host for several months.

To the above general scheme there are several exceptions, for example—

In *R. bursa* the metamorphosis of the larva into pupa takes place on the host, after which the pupa falls to the ground for its final transformation.

In *Margaropus annulatus* both the larva and pupa undergo their changes on the same host.

H. ægyptium is said to be parasitic only in the adult form.

Ornithodorus moubata passes the larval stage within the actual egg, the pupa emerging on rupture of the shell.

Ticks, both immature and adult, can withstand starvation for some months, or, as adults, even for years, provided that their environment be of the necessary humidity.

Their longevity varies from three weeks (*Margaropus annulatus*) or eleven weeks (*R. appendiculatus*) to some years. If there is no lack of food the duration of life is short: starvation arrests growth, and prolongs life considerably.

The *fertility* of ticks is very great, the female, at a single laying, often producing as many as 20,000 eggs.

Ornithodorus moubata is, however, much less fertile and lays less than a couple of hundred eggs. This pecularity is probably due to a diminished destruction of eggs or larvæ by natural enemies, owing to the peculiar habits of the adult. The habits of this tick somewhat resemble those of the ordinary bed-bug, for it hides in crevices of native huts during the day and comes out at night to suck blood.

Infection.—Texas fever of cattle can be transmitted through the larvæ hatched from the ova of *Margaropus annulatus* if the female is previously fed on infected blood.

In *R. bursa* only the mature tick can so transmit the disease.

Anatomy.—The dorsum is membranous, or chitinous, or with chitinous anterior plate.

The *eyes* are simple.

The *capitulum* is a movable sclerite in front of the 1st pair of legs, and bears the mouth parts. These latter consist of:—

The *labium* (or *hypostoma*)—a median piercing probe, with recurved teeth.

The *mandibles* (or *chelicerœ*), which are a pair of slender organs

lying dorsally to the labium. The terminal portion has two or three apophyses bearing recurved teeth. The mandibles are encased in the *mandibular sheath*, a finely toothed membranous sheath —forming, with the mandibles and labium, the piercing organ, or *haustellum.*

The *palpi* are four-jointed and serve as a kind of sheath for the haustellum.

The *legs* have six segments—coxa, trochanter, femur, patella, tibia, and tarsus. The tarsus is slender, and bears two claws, and sometimes also a membranous sucker beneath them.

The anterior tarsus bears *Haller's sense organ* near its apex.

The *stigmata* are situated laterally and ventrally behind the level of the 4th leg in *Ixodidæ*, where they open into a peritreme or stigmal plate. In the *Argasidæ* they lie between the 3rd and 4th legs.

The *genital orifice* is in the middle line of the ventral surface, a little behind the rostrum.

The *anus* is valvular, and is situated near the posterior ventral margin. In the males of some species this is surrounded by four *anal plates* or *clypei.*

Classification.

1.—Fam. Argasidæ.

The *scutum* is absent.

The *capitulum* is concealed by a non-chitinous extension of the dorsal area.

The *integument* is uniformly granular.

The *palpi* are unmodified, the 4th segment being non-retractile; the 1st and 4th, long; and the 2nd and 3rd not excavated.

There is no *claw sucker* in the adult.

Sexual dimorphism is slight. They are often found about houses; are frequently parasitic on domestic poultry. They hide in the day and feed at night.

Table of Genera (after *Pocock*).

A.—Body with a lateral cariniform edge, of different sculpture to rest of dorsum or venter.

(1) With a deep post anal integumental groove.

Genus:—*Caris*.

(2) Post anal groove absent.

Genus:—*Argas*.

B.—Body not laterally carinate; sculpture as that of dorsum.

(1) With a movable sclerite on each side of palpi.

Genus:—*Alectorobius*.

(2) No movable sclerite.

Genus:—*Ornithodorus*.

Notes.—1. Genus *Caris* (Latrielle).—Mistaken for *Argas* by some authors. Type species is *C. vespertilionis*, a parasite of European bats. The body is as wide as long.

2. Genus *Argas* (Latreille).—Body is hollowed or flat dorsally, and oval in shape. The eyes are absent. There are eleven species, one of which transmits the "Spirillar fever" of poultry.

A. reflexus is a parasite of European pigeons.

A. persicus is the Garib-Guez of Persia, giving a severe bite.

A. tholozani, of Persia, also has a malignant bite.

3. Genus *Alectorobius* (Pocock)—Type species. *A. Talaje* (Guérin), known in Mexico and Columbia as "chinche," where it is a great pest. Represented in Europe by *A. coniceps*, and in S. Africa by *A. capensis*.

4. Genus *Ornithodorus* (Koch).—Eyes present or absent. The species *O. moubata* is common in W. Africa, and is the intermediate host which conveys "Spirillar (or tick) fever."

II.—Fam. Ixodidæ.

The *scutum* is present.

The *capitulum* is exposed, and projects at the anterior end of the body.

The *palpi* are modified. The 1st segment is short; the 2nd and 3rd longer and excavated internally; and the 4th minute and retractile.

There is a *claw-sucker* in the adult.

Sexual dimorphism is pronounced.

♂ The tergum is covered with a dorsal plate. There are ventral sclerites.

♀ The dorsal plate is only on the anterior part. There are no ventral sclerites. The integument is highly distensible.

TABLE OF GENERA.

I. Sub-family *Ixodinæ.*—*Males* (*a*) Dorsal integument thickly chitinised throughout. No porous areas on capitulum.

(*b*) Ventral surface strengthened by five or seven chitinous plates grooved as in the females.

Females (*a*) Dorsal surface with small chitinous plates at the anterior extremity; and two porous areas on the capitulum.

(*b*) Transverse, recurved pre-anal groove, surrounding the anus and extending back to the posterior edge of the body.

(i.) Third segment of palpus long and acuminate.
Genus:—*Ceratixodes.*

(ii.) Third palpal segment short with rounder apex.

(*x*) Palpi not excavated internally.
Genus:—*Eschatocephalus.*

(*y*) Palpi excavated internally.
Genus:—*Ixodes.*

II. Sub-family *Rhipicephalinæ*.—*Males* (*a*) As in *Ixodinæ*.

(*b*) Ventral surface soft; or with two or four adanal plates.

Females (*a*) As in *Ixodinæ*.

(*b*) No recurved pre-anal groove. The post-anal groove meets the two long grooves diverging backwards from the genital orifice.

(i.) Palpi long and slender.

(*x*) No eyes.

Genus:—*Aponomma*.

(*y*) With eyes.

Genus:—*Hyalomma*.
Amblyomma.

(ii.) Palpi short; 2nd segment as wide as long.

(*x*) No eyes.

Genus:—*Hæmaphysalis*.

(*y*) Eyes.

α. Capitulum transversely oblong ♀. Fourth coxæ enormously enlarged ♂.

Genus:—*Dermacentor*.

β. Capitulum hexagonal ♀.

(*a*) Spiracular area sub-circular. Pre-anal groove obsolete.

Genus:—*Margaropus*.

(*b*) Spiracular area comma-shaped. Pre-anal groove present.

Genus:—*Rhipicephalus*.

Notes.—(After *Pocock*, *Braun*, and *Stephens*)—1. Genus *Ceratiscodes* (Neumann, 1902).—*C. putus* is the only known species of this genus. It is parasitic on certain sea-birds.

2. Genus *Eschatocephalus* (Frauenfeld, 1853).—Several species found in caves as parasites of bats.

3. Genus *Ixodes* (Latreille, 1795).—Are small sized ticks. The genus contains a large number of species, none of which are known to be pathogenic. They are parasitic on horses, cattle, deer, hedgehogs, moles, bats, birds, and lizards.

Two well-known species are:—

I. ricinus—the castor-bean tick, common on cattle in Europe.
I. hexagonus—the European dog tick.

4. Genus *Aponomma* (Neumann, 1899).—This is an exotic genus containing very few species. It is parasitic on tropical reptiles.

5. Genus *Hyalomma* (Koch, 1879).—Contains only a small number of species; parasitic on certain mammals and on tortoises.

6. Genus *Amblyomma* (Koch, 1844).—There are over eighty species; parasitic on mammals.

A. variegatum is frequently found on cattle in Rhodesia.

7. Genus *Hæmaphysalis* (Koch, 1844).—There are no eyes. The 2nd palpal segment has a lateral spine. Coxa i. not bifid in either sex. Coxa iv. normal in ♂.

The genus contains about twenty-six species, of which *H. leachi*—the S. African dog tick—possibly transmits Piroplasma canis. This species is also found on cattle.

8. Genus *Dermacentor* (Koch, 1844).—Eyes are present. The palpi are short and thick. Coxa i. is bispinate in both sexes. Coxa iv. enlarged in the male.

About twenty-four species are known.

D. electus is the American dog tick.

9. Genus *Margaropus* (Karsch).—Formerly known as "Boophilus." There is only one known species, the *M. annulatus* (say), which transmits the Texas fever of cattle.

10. Genus *Rhipicephalus* (Koch, 1844)—(= *Eurhipicephalus* of Neumann).—Possesses eyes. The capitulum is hexagonal. The 3rd palpal segment has a recurved spine. The male has one or two pairs of adanal plates.

There are more than thirty known species, mostly African—and some pathogenic.

R. annulatus	transmits	American	Texas fever.
R. decoloratus	,,	S. African	,,
R. australis	,,	Australian	,,

CHAPTER XI.

SNAKE AND OTHER VENOMOUS BITES.

POISONOUS bites or stings occur chiefly amongst members of three of the zoological sub-kingdoms.

In the subjoined list, those orders have been marked with an asterisk to which the harmful genera and species belong :—

Sub-kingdom	Class.	Order.
Sub-kingdom *Cœlenterata*	I. *Hydrozoa*	1. *Hydrida* (Polypes).
		2. *Corynida*.
		3. *Thecaphora*.
		4. *Trachymedusæ* (jelly fish).*
	II. *Actinozoa*	1. *Zoantharia* (sea anemones; corals).
		2. *Alcyonaria* (red coral).
		3. *Rugosa* (coral).
		4. *Ctenophora*.
Sub-kingdom *Arthropoda*	I. *Crustacea*.	
	II. *Arachnida*	1. *Podosoma* (sea spiders).
		2. *Acarina* (mites and ticks).*
		3. *Pedipalpi* (scorpions).*
		4. *Araneida* (spiders).*
	III. *Myriapoda*	1. *Chilopoda* (centipedes).*
		2. *Chilognatha* (millipedes).
		3. *Pauropoda*.
	IV. *Insecta* .	1. *Hemiptera* (aphides, bugs, and licades).*
		2. *Aphaniptera* (fleas).*
		3. *Diptera* (flies and gnats).*
		4. *Lepidoptera* (butterflies and moths).*
		5. *Hymenoptera* (ants, wasps, and bees).*
		6. *Coleoptera* (beetles).*
Sub-kingdom *Vertebrata*	I. *Pisces* .	1. *Pharyngobranchii* (lancelet).
		2. *Marsipobranchii* (lampreys).
		3. *Teleostei* (bony fish).*
		4. *Ganoidei*.
		5. *Elasmobranchii* (sharks and rays).*
		6. *Dipnoi* (mud fish).
	II. *Amphibia*	1. *Ophiomorpha*.
		2. *Urodela*.
		3. *Anoura* (frogs and toads).*
	III. *Reptilia* .	1. *Chelonia* (tortoises and turtles).
		2. *Ophidia* (snakes).*
		3. *Lacertilia* (lizards).
		4. *Crocodilia* (crocodiles and alligators).

The following remarks give the chief points in connection with ıe bites or stings of the various species :—

Jelly-fish.—Some medusæ or jelly-fish are poisonous when they ome in contact with the skin while bathing or swimming.

The *poison* apparatus is contained in surface tubercles, and the oison is transmitted by contact—*e.g.*, *Cyanea capillata* and *Physalea elagica* (or Portuguese man-o'-war).

The *manifestation* is a local stinging pain, followed by the formaon of urticarial weals.

The *treatment* is best obtained by the application of Carron oil ocally. One of the best ointments possible for this or like conitions is :—

℞.—Adip. Lanæ,	℥i.
Ung. Paraffin,	℥iij.
Ol. Amygdal.,	℥iv.

Warm, and add while stirring—

Liq. Calcis,	℥i.
Liq. Plumbi Fort,	ℨss.
Ol. Caryoph.,	♏v.

This is much superior to Carron oil.

Ticks.—The order of Acarina contains many biting ticks which ttack animals. Ticks have a predilection for certain hosts, but the ırval and adult stages may be passed on different animals.

The chief species that attack man are :—

1. *Leptus*, in C. America and West Indies.
2. *Tetrarhynchus irritans*, in Mississippi Valley.
3. *Argas Persicus*, common in Persia.
4. *Ornithodorus moubata*, found on the Zambesi.
5. *Alectorobius talaje*, of Guatemala.

No. 1 is a hairy tick, and occasionally burrows in the skin.

No. 3 is of a blood-red colour, with yellow feet, and spotted with hite on the back.

No. 4 is the cause of tick fever in Uganda.

No. 5 is nocturnal in habit and lives in holes of bamboo walls, esembling *Cimex* in many ways.

Symptoms.—There is local œdema, irritation, and itching, and ccasionally urticaria. A certain amount of constitutional disturbnce is also produced at times.

Treatment.—Antiseptic evaporating lotions.

The subject is dealt with at length in Chapter x.

Scorpions.—These members of the Pedipalpi order are found ı all tropical countries, varying in length from 2 to 6 inches; enerally nocturnal in habit. They possess a segmented abdomen, ıe last six joints being narrowed into a tail, at the extremity of hich is a curved hook, in which are two orifices. There is a gland eceptacle at the base of the tail, and a duct conveys the poison

to the openings in the hooked end. Their habitat is largely in rotten wood.

The *character* of the sting is very like, though more severe than, that of the wasp.

As *treatment*, the application of ammonia or an evaporating lotion to the affected part will usually be sufficient, though occasionally the internal administration of stimulants may be indicated.

It has also been recommended to incise the wound and rub in crystals of potassium permanganate, as in the case of snake bite.

Spiders.—A few of the Araneida order can inflict poisonous bites on man, especially some of the larger tropical forms.

The poison apparatus consists of an elongated poison sac, communicating with the exterior through a fissure near the point of the hard curved fang which constitutes the last joint of the falces or modified mandibles.

The *symptoms*, however, which follow the bite of most spiders are seldom more than little or much local irritation, though the bite of some, such as *Lycosa tarantula* is said to cause extreme symptoms.

The bite is nevertheless immediately fatal to the many small animals which form the normal diet of the spider.

Nothing is needed by way of *treatment* except soothing local applications; although in case of some tarantula bites, stimulants will be necessary.

Centipedes.—These examples of the Chilopoda order are ubiquitous in the tropics—largely, but not invariably, nocturnal in habit. They frequent stables, and enter houses frequently, a favourite place of repose being inside boots or shoes. They are usually about 4 inches long, but may attain a length of 8 inches. The poison is at the head, not at the tail as in the case of scorpions.

The mandibles (like a pair of dilated feet) have hooked points with an aperture for ejecting the poison.

The *symptoms* consist in considerable local irritation, and frequently slight constitutional disturbance.

The poison is probably identical with that of the scorpion.

As *treatment* the same methods should be adopted as recommended for scorpion bites.

Bugs.—These are insects belonging to the order of Hemiptera.

They have a suctorial mouth, with a grooved rostrum for piercing.

They have a distinct and offensive smell. The most well known member is the familar *Cimex lectularius* or bed bug which is ubiquitous.

An even more obnoxious specimen is the Benchuca, or great black bug of the pampas of S. America.

The *symptoms* following a bite are too well known to need description. The irritation and localised induration are more intense and persistent than the bites of the Aphaniptera.

They may act possibly as intermediate hosts for parasites or bacteria, and it seems not unlikely that they may have something to do with the spread of leprosy.

As a rule, no *treatment* is required.

Fleas.—The fleas, or members of the Aphaniptera order, are niversal parasites of warm-blooded animals. An account of their fe-history and classification will be found in Chapter x.

As a rule, the different species of fleas affect a special host, and ney do not attack another kind of host unless driven by hunger.

The common flea of man is *Pulex irritans*. *Symptoms*—The bite irritating, but not severe or peristent.

One species, the *Sarcopsylla penetrans* or jigger, is very common in ne West Indies. The male is parasitic to the pig. The female, hen impregnated, will attack any warm-blooded animal, especially an, and burrows into the skin, causing local irritation and often us formation. The eggs are thus discharged to the surface.

In the matter of *treatment*, none is required for the ordinary flea te. An encysted chiggœ (or jigger) should be enucleated with a eedle and the cavity touched with neat carbolic acid.

Diptera.—The blood sucking diptera in the tropics are many. he chief groups are the following:—

1. *Culicidæ*.—Comprising the mosquitoes and gnats. Notes on eir life-history, habits, &c., will be found in Chapter x.

2. *Chironomidæ* or midges.—These are widely distributed in e tropics. A very minute specimen, the *Ceratopogon*, is an ceptionally persistent blood sucker. He can get through the eshes of most mosquito-nets, and the bite, though hardly felt at e time, will irritate for days, especially when warm in bed. They e commonly called sand flies in the West Indies.

3. *Psychodidæ* or moth flies.—These are also very small and e found both in Europe and the tropics.

The best known is *Phlebotomus*. The bite is not as irritating as at of the *Chironomidæ*.

4. *Simulidæ* (sand-flies or buffalo gnats).—These are small impbacked flies. Antennæ, eleven segments, destitute of hairs. lpi, four segments.

Proboscis has epi- and hypo-pharynx and is short and thick. Wings e relatively large. They are not very fierce and the bite is not ry irritating.

5. *Tabanidæ* (horse or gadflies.)—There are some 1,300 species, any of which are found in the tropics.

The male fly does not bite. No special features can be mentioned to the female bite, which resembles that of other flies.

6. *Glossina* (Tsetse flies.)—These are well distributed in the opics. The bite itself is not a matter of much importance, but veral species act as hosts in the spread of *Piroplasma bigeminum* d of some Trypanosomes. These are:—*G. fusca*; *G. morsitans*; d *G. pallidipes*.

Lepidoptera.—The majority of butterflies and moths are harmss. The hairs of the larvæ of some species have, however, isonous properties. "In Ceylon, a greenish hairy caterpillar, ngitudinally striped, probably of the genus *Bombyx*, which

frequents the leaves of the *Hibiscus*, alighting on the skin causes as much irritation as the sting of a nettle.

"The larvæ of *Neæra lepida* have similar properties. They are short and broad, of a pale green, with fleshy spines on the upper surface, each of which is charged with venom that occasions acute suffering. The larvæ of *Adolia* are also armed with venomous hairs. Another, not uncommon in certain trees in terai of the Himalaya, is a dark coloured hairy caterpillar, which is apt to fall on people and cause intense irritation. It is known as 'Komlah,' but the imago is not known" (*Fayrer*).

Hymenoptera.—*Ants* are ubiquitous. The act of biting with their mandibles extrudes the poison, which consists of formic acid.

The largest known ant, called *Camponotus*, a red and black insect reaching 1¼ inches in length gives a powerful bite, but the most vicious is that of the Caringa (*Æcophylla smaragdina*), a red ant which builds nests in trees by cementing the leaves together. The irritation from a bite may last for several days.

Bees and Wasps.—These are represented in the tropical fauna, but call for no special mention. They are certainly not as fierce as their brethren in the colder climates, nor do wasps seem so attached to fruit, possibly because they can get a surfeit in the jungle without coming to the haunts of man.

An occasional specimen may prove an unwelcome addition to a luscious mouthful, and the resulting sting in the throat may cause dyspnœa or even œdema of the glottis, necessitating an emetic, and very likely tracheotomy.

The poison sac and sting are in the tail.

In some hymenoptera, such as *Ophion* and *Paripla*, the female ovipositor is used also as a weapon.

Coleoptera.—Several members of this order (beetles) have an acrid secretion which excites inflammation or vesication. Examples of these are:—

Mylabris cichorii, in India.
Cantharis, in Senegal.
Lytta vittata, in America.
Lytta ruficeps, in Chili.

The same *treatment* as in the case of jelly-fish stings should be adopted.

Pisces.—The fish have no poison glands analogous to those in the other natural orders.

Many, however, such as the Rays, are armed with sharp or serrated opercular or fin spines, which can inflict lacerated and painful injuries.

In several genera (*e.g.*, *Thalassophryne*) there is a distinct receptacle or sac for the irritating fluid. In others (such as *Trachinus* or weever) there is a cavity in the spine itself.

The *treatment* should take the form of sedative fomentations, and, if there is much depression, alcohol should be administered.

Amphibia.—There is no poison apparatus in Amphibia like that the *Ophidia.*

Toads and Salamanders, however, have certain glands in the back hich secrete a venomous fluid. Thus if a toad be seized by a dog, uch local irritation and salivation may be caused in the dog's outh.

The glandular venom, if injected into guinea-pigs and other ıimals, causes cerebro-spinal effects, and death results.

Reptilia.—The poisonous biters of this class are confined to one ·der—the **Ophidia** or snakes.

They are abundant in the tropical zone, and comprise nearly 2,000 ıown species.

All the snakes are carnivorous. As a rule, they do not resent ›proach, and only a few poisonous species, such as *Ophiophagus ıps*, will follow and attack a human being. We may class the ıakes in five categories :—

1. **Ground Snakes.**—Body cylindrical, flexible, with smooth or keeled scales. Food, chiefly terrestrial vertebrates. Majority non-poisonous. Comprise, however, the most poisonous of all. Oviparous (except the vipers).
2. **Tree Snakes.**—Body compressed and slender. Ventral scutes carinate on sides. Tail prehensile. Eye large. Colour, often bright. Food, arboreal animals. Species, both poisonous and innocuous. Oviparous.
3. **Sea Snakes.**—Compressed rudder-shaped tail. Fifty species known. Inhabit Indo-Pacific Ocean. Completely marine, cannot move on land. Food, fishes. Viviparous and poisonous.
4. **Fresh-water Snakes.**—Body cylindrical. Tail tapering. Head flat. Viviparous and innocuous.
5. **Burrowing Snakes.**—Cylindrical rigid body. Teeth few. Short strong tail. Rudimentary eye. All innocuous. Oviparous.

The following are the more important of the poisonous snakes :—

1. *Naja tripudians* (*cobra*), 5 to 7 feet long. Distrib.—India, China, E. Indian Archipel., Africa, &c. Responsible in India for about 200 deaths per mille.
2. *Crotalidæ* (*rattlesnakes*), 3 to 5 feet. Distrib.—N. and S. America.
3. *Vipera Russellii* (*Tic Polonga*), 4 feet. Distrib.—India, Burmah, and Ceylon. Is very deadly.
4. *Ophiophagus elaps* (*Hamadryad*), 14 feet. Geogr. range. Identical with cobra, but is scarcer and more dangerous.

5. *Boa constrictor*, 7 to 10 feet. Distrib.—Central America to Brazil.

6. *Boa murina* (*anaconda*), 20 to 30 feet. Distrib.—S. America and Trop. Pacific.

7. *Trigonocephalus* (*pit viper*), 6 feet. Distrib.—Central America and West Indies. Eats rats on sugar plantations, &c.

Anatomical Features of Snake Skulls.—The skull is well ossified and the cranial bones distinct.

The mandible (lower jaw) is complex and united to the skull by movable quadrate and squamosal bones. It may have many or few teeth. Its symphysis is loose. The maxillæ (upper jaw) is sometimes long with numerous teeth, or may be shortened with a few teeth or only one tooth. In the poisonous snakes the poison fang is situated in the maxilla; this is a long conical tooth with a groove on the anterior aspect communicating with a compound racemose gland in the temporal region. The gland secretes a clear viscid fluid like glycerine, and when the mouth is closed, this fluid is ejected by muscular action along the groove of the fang.

In the *Crotalidæ*, opening of the mouth rotates the fang vertically; when the mouth is closed the fang is horizontal. In the genus *Naja*, the fang is not capable of erection.

The pterygoid and palatine bones also carry teeth, some more and some less.

Physiology of Snake Poison.—The poison gland can be regarded as a modified salivary gland, and is situated below and behind the eye.

The venom looks like glycerine; has an acid taste, and coagulates with heat. Its action is most active on warm-blooded animals. It is most rapid when injected into the blood, but can also be absorbed by mucous and serous membranes.

Oppenheimer recognises four principles in snake poison:—

1. Hæmorrhagin.
2. Hæmo-agglutinin.
3. Hæmo-lysin.
4. Neurotoxin.

No. 1. Is the chief constituent of rattlesnake venom. It attacks the endothelium of vessel walls. It is a globulin, obtained as a white precipitate by dilution; easily redissolved in saline solution, and is then very toxic.

No. 2. It attacks the red blood-corpuscles. It is destroyed by a temperature of 170° F.

No. 3. Is the chief constituent of cobra poison. It attacks the red blood-corpuscles. Its local effects are slight, but it is rapidly absorbed. It is a peptone.

No. 4. Attacks the cells of the central nervous system.

Poisonous snakes are found to be immune to the venom of other snakes of their genus.

Snake-poison Antivenenes.—1. Calmette's antivenene serum. Made at Lille Pasteur Institute. Cobra venom employed.

2. Brasil's anti-ophidic serum. Made in Caracas. Rattlesnake venom employed.

Remarks.—A commission reported on the therapeutic value and comparison of the above in January, 1906. They found that three-tenths of a milligram of rattlesnake venom was fatal to a guinea-pig. They, therefore, took two guinea-pigs and injected 5 c.c. of Calmette's serum into one and 5 c.c. of Brasil's serum into the other. Then half a milligram of rattlesnake venom was injected into both. The former died in six hours, the latter showed no ill effects.

It seems certain, therefore, that the composition of the venoms varies too much in different snakes to be able to employ a single antivenene.

Calmette's should be used to protect against cobra bites and Brasil's against rattlesnake bites.

Clinical Features of Snake-bites.—The *degree of danger* depends on:—1. Quantity of poison injected (worse in case of large and vigorous snakes).

2. Strength of individual bitten (worse in children or weakly persons).

3. Position and depth of bite (worse in vascular tissue or veins).

The *local effects* are:—Pain, swelling, and paresis; also (if not death) cellulitis and sloughing.

The *general effects* are:—Fainting, depression, nausea, dyspnœa, vomiting, loss of co-ordination, relaxed sphincters, coma, and convulsions.

The constitution of the blood is altered: the nerve centres are paralysed. Death occurs by asphyxia.

Post-mortem, the blood is found very fluid.

The *prognosis* is usually a matter of prompt and early treatment, which gives the only possibility of recovery if the bite has been by one of the more poisonous snakes.

Treatment of Snake-bite.—The treatment summarised by *Günther* as long ago as 1864 can hardly be improved upon:—

1. If on extremities, ligature tightly above the wound.
2. Freely enlarge the punctured wounds.
3. Suck the wound (if no abrasion in mouth).
4. Cauterise with hot iron or $AgNO_3$.
5. Inject Pot. permang. into the wound.
6. Give stimulants internally.

The only difference in modern procedure is that devised by *Lauder Brunton*, where, instead of 3, 4, and 5 above, the enlarged wound is packed with permanganate crystals, and well rubbed in.

Ferris & Co. of Bristol sell a first-aid snake-bite outfit, consisting of a small box containing a lancet on a hollow wooden stem con-

taining pot. permanganate crystals; also a small bottle of crystals, tape, lint and plaster. The whole weighs $2\frac{1}{4}$ ounces and measures $1\frac{3}{4}$ inches by 1 inch, costing 3s. 6d.

Rogers records twenty recoveries out of twenty-one cases, in India, under this treatment. Thirteen of the snakes were identified, being mostly cobras. The one death was in the case of a woman not treated until eleven hours after the bite, and then moribund.

So many lives are sacrificed by snake-bite in India, every year, that Messrs. Arnold & Sons have made, at Lauder Brunton's suggestion, a cheap lancet, mounted in a hollow wooden case to contain permanganate of potash crystals and priced at 6d each, with the hope that they might be placed on sale at post-offices throughout that country in the same way that packets of quinine are now sold.

CHAPTER XII.

ANKYLOSTOMIASIS.

Definition.—An anæmic cachexia induced by the invasion of the human intestinal canal by a nematode, the *Ankylostoma duodenale* (Dubini, 1843).

Synonyms.—I. Of the parasite:—*Strongylus quadridentatus* v. Sieb., 1851; *Dochmius ankylostomum*, Molin, 1860; *Sclerostoma duodenale*, Cobb., 1864; *Strongylus duodenalis*, Schneid, 1866; *Dochmius duodenalis*, Leuckart, 1876.

II. Of the disease:—*Egyptian chlorosis; Tropical chlorosis; Ceylon anæmia; Miners' anæmia; Malcœur: Cachexie aqueuse: Tuntum* (Colombia).

History and Geographical Distribution.—Apparently the first report of the disease was in 1864 when *Piso* reported a disease "Oppilatio" from Brazil. This must be certainly the Latin derivation of the term "oppilacao" by which the disease is now called in that country.

Dubini in Milan was the first to discover the nematode, in 1838.

Pruner found it in 1846; and soon afterwards *Bilharz* discovered it in Egypt; where *Griesinger* recognised it as the cause of "Egyptian chlorosis."

Considerable attention was called to the disease, owing to its epidemic appearance amongst the staff engaged in the construction of the St. Gothard's tunnel.

Perroncito demonstrated that the condition known as "Miners' anæmia" was due to the same cause.

The distribution of *A. duodenale* is world-wide, but it is chiefly in tropical countries that it is endemic.

Endemically it is found in Egypt, Abyssinia, Tunis, Algiers, Madeira, the West Coast, Cape Colony, Natal, Orange River Colony, Zanzibar, Madagascar, Mauritius, India, Assam, Ceylon, Malay Peninsula and Archipelago, Philippines, Japan, Southern States of America. Central America, most of South America, Australia, New Guinea, Fiji. It is also very prevalent amongst certain labourers, bricklayers, and miners in certain European districts.

Natural History.—The parasite belongs to the order of *Nematodes*, the family *Strongylidæ* and the genus *Ankylostoma*. (Figs. 23 to 27).

The body is cylindrical and of a light reddish colour. In the

mouth will be seen four hooked teeth ventrally situated and two dorsally.

The *males* measure 6 to 8 mm. in length (sometimes 10 mm.); and in breadth are 0·4 to 0·5 mm. The body terminates in a bell-shaped bursa, which has lateral and dorsal alar processes supported by ribs. There is a bottle-shaped penis and two long thin spicules.

The *females* measure 10 to 12 mm. in length (sometimes 18 mm.); and in breadth are 1 mm.

The caudal extremity has a small spine. The vulva is in the anterior part of the posterior quarter, and leads to a double muscular vagina, communicating with an anterior or posterior uterus and a tortuous ovary.

In copulation the worms resemble in outline the Greek letter γ; the male grasping the female by means of the bursa, fixed by the introduction of the spicules into the vulva.

The *ova* (Fig. 28) are elliptical, with thin shells which are divided from the segmented yolk by a transparent fluid. They measure 60 μ in length by 40 μ in breadth. No development takes place in the bowel of their host, but they are evacuated with the fæces in the same state of segmentation in which they are laid, the reason being found in the lack of oxygen.

Habitat.—A. duodenale lives in the duodenum, more rarely in the jejunum. Dubini, however, considered the jejunum as the favourite site.

The worm fastens on to the mucous membrane by means of its oral capsule and teeth. The pointed process at the capsular base is then employed to open the blood-vessels and suck the blood. The nutrition of the worm is derived from the plasma, since the corpuscles are evacuated unchanged.

Many hundreds and thousands of parasites may be found in the intestine, the females preponderating in the proportion of about 3 to 1. This nematode was found by *Levaillant* in the gibbon; and by *Leuckart* in the gorilla.

Development.—After being launched on the world with the fæces, the segmented ova begin to develop if the temperature happens to be sufficiently high (20° to 25° C.).

This development takes place best in fæcal matter, and will not proceed at all in pure water. The embryos develop in one or two days into rhabditiform larvæ, measuring 0·21 mm. in length, and burst through the end of the egg-shell, exhibiting lively movements.

In a few days they grow to about 0·6 mm. and moult; a second moult follows very shortly, and the pharyngeal bulb disappears. Nevertheless they still retain their larval integument and remain motile. Now, however, they are no longer sensitive to water, and can be kept alive in it for three or more months.

Giles states that he was able to hatch out embryos on sand from ankylostome ova, and that he obtained adults from these, which were different from the ordinary intestinal species. This would seem to show that the parasite may have an extra corporeal life cycle,

Fig. 23.

Fig. 24. Fig. 25.

Fig. 26.

Fig. 27.

Fig. 23. Ovum, 60 μ × 40 μ.
" 24. A. duodenale (male), 8 mm.
" 25. A. duodenale (female), 12 mm.
" 26. A. duodenale (natural size).
" 27. Method of sexual congress.

Fig. 28. Ovum of *Ankylostoma duodenale* (× 500).

with possibly two different forms of development, as in the case of *Rhabdonema strongyloides.*

Etiology.—Neither sex nor age will confer immunity. Even small infants may be attacked. There is no racial predisposition.

Transmission cannot take place by dust, since larvæ and ova cannot withstand desiccation.

Infection is brought about in one of two ways:—

1. By the mouth.
2. By the skin.

1. *By the Mouth.—Looss'* experimental infections of dogs and cats have shown the subsequent development. The larvæ do not stay long in the stomach. They moult on the 7th day and again on the 15th day, by which time an oral capsule is formed, and the alimentary and genital systems elaborated to some extent. They are then ·9 mm. long. In four or five weeks maturity is attained, and copulation takes place.

2. *By the Skin.—Looss* discovered this second means of entry.

After the first or second exogenous ecdysis, the embryo can penetrate the skin (usually of the feet or legs of coolies working on contaminated soil). This it does through some follicle, and thence into a blood-vessel, reaching the lungs *via* the arteries and heart. Here it leaves the blood-vessels, enters an air vesicle, and then travels *via* bronchus, trachea, œsophagus, and stomach till it reaches its normal habitat. Arrived there, sexual characters are developed and coitus takes place.

In their passage through the skin a local dermatitis is not infrequently set up—which has for long been known as "*Coolie itch*" or "*ground itch*" without anyone suspecting it to be merely a phase of Ankylostomiasis.

Symptoms.—A moderate infection may cause no symptoms at all. Probably several hundred are necessary to cause damage to the general health.

The signs and symptoms are:—

1. Ova in the fæces.
2. Anæmia.
3. Digestive disorders.

The *dyspeptic troubles* are the result of chronic irritation of the intestinal tract, and are generally manifested by epigastric pain and uneasiness, increased by pressure and temporarily relieved by food. Colic, flatulence, and diarrhœa may be present.

The *anæmia* is in part due to the large extraction of blood, but also to the elaboration of a toxin by the parasite, causing hæmolysis and eosinophilia

"Charcot-Leyden's crystals" (probably a product of the parasite) are frequently found in the stools.

Palpitation of the heart is not infrequent. The pulse is accelerated and sometimes irregular. Hæmic bruits may be present.

The *skin* is livid, pallid, and rather yellowish (as in malaria or cancerous cachexia).

The *mucous membranes* are pale.

The *urine* is copious and pale, but rarely albuminous.

The *temperature* is usually normal or sub-normal; but at the commencement of the disease, occasional irregular fever may occur.

Retinal hæmorrhages are sometimes observed.

Headache, giddiness, weakness, and apathy may all be found in advanced cases.

There may be spontaneous recovery; otherwise if the parasites be not expelled, death from exhaustion, dropsy, or diarrhœa may take place, or the patient die from intercurrent disease.

Pathology and Morbid Anatomy.—There is bodily emaciation and pallor of most organs. The *lungs* are usually œdematous. The left *heart* is frequently hypertrophied.

Daniels has found grains of yellow pigment (with hæmatoidin reaction) in the parenchyma cells of liver and kidneys.

The stomach usually shows signs of chronic catarrh. The jejunum is the seat of numerous petechiæ.

In fresh cases, the mucous membrane is thickened; and the small intestine may contain sanguineous or chocolate coloured contents.

Diagnosis and Prognosis.—The discovery of Ankylostome eggs in the stool of a patient will clear up the diagnosis.

Chlorosis, pernicious anæmia, malarial or cancerous cachexia have some symptoms of a like nature. These diseases should therefore be eliminated.

Under treatment, the prognosis is quite favourable. *Sandwith* reports the cure or relief of 89·5 per cent. of about 400 cases which had come under his observation in Egypt.

Treatment.—Oil of peppermint, kerosene, guaiacol, &c., have all been recommended, but do not accomplish the desired purpose.

The best drug is *Thymol.* The patient should be put on liquid diet for a day or two, and the bowels well evacuated. Then three or four doses of thymol (gr. xx.) are given on an empty stomach at intervals of an hour.

One such course may suffice to dislodge all the parasites; but it is better to examine the stools for ova after a week, and, if necessary, repeat the course.

Precautions should be taken when using Thymol:—

1. Lie down after doses for some hours.

2. Avoid alcohol, ether, glycerine, chloroform, &c., which are all solvents of thymol, and may cause undue absorption with toxic symptoms.

3. Do not give in advanced cases, with much prostration.

4. Gastritis, dysentery, nephritis, and morbus cordis are all contra-indications for its exhibition.

Male-fern is sometimes of use. It should be given as in tapeworm treatment. A preliminary purge is followed by 0·5 of

Extr. filicis liq. in gelatin capsules, six such capsules being given = ʒi.).

Two hours later, a saline aperient should be administered.

Castor-oil should be avoided, as the poisonous filicic acid is soluble in the oil and toxic symptoms may follow.

Pelletierine tannate may also be tried.

℞. Pelletierinæ tannatis . . . gr. viii.

Sig.—Should be taken fasting, and followed in two hours by ol. ricini, ʒi.

Prophylaxis.—The two modes of infection should be borne in mind.

1. (*a*) The water supply should be guarded from fæcal contamination.

(*b*) Drinking water boiled and filtered.
(*c*) Vegetables washed and cooked.
(*d*) Dishes and utensils well cleaned.

2. (*a*) Fæcal contamination of the soil prevented.

(*b*) Accessible privy accommodation provided.
(*c*) Promiscous defecation prohibited.
(*d*) Stools disinfected.
(*e*) Cases discovered and treated.

ANKYLOSTOMA AMERICANA.

(*Uncinaria Americana*, Stiles.)

In May, 1902, Stiles discovered that Ankylostomiasis in America was not, as a rule, due to *A. duodenale*, but to the above new species.

The two species can readily be distinguished.

A. Americana is shorter and more slender. The male, 7 to 9 mm. by ·3 mm. The female, 9 to 11 mm. by 0·4 mm.

The buccal capsule is smaller, and has semilunar plates instead of hooked teeth.

The eggs are larger than those of *A. duodenale*, being 64 to 75 μ long, by 36 to 40 μ broad.

It has been found, so far, only in man; and the life history has not been investigated.

CHAPTER XIII.

BERI-BERI.

Definition.—A specific multiple neuritis, characterised by liability to implication of the phrenic and pneumogastric nerves, and by occurrence of serous effusions.

It is endemic in certain tropical regions, and may appear in epidemic form in institutions such as gaols, coolie lines, &c.

Synonyms.—The word beri-beri is officially recognised by the London College of Physicans. The origin is undetermined, but is certainly Eastern. The following derivations for the term have been suggested :—

WORD.	LANGUAGE.	AUTHORITY.
Beri (a sheep).	Hindustani.	Meyer-Abrens.
Bharyee (weak movement).	Singhalese.	Marshall.
Bharbari (swelling).	Hindustani.	Herklots.
Biribi (stiff walking).	Sudanese.	Plateeuw.
Buhr (asthma). *Bahri* (a sailor).	Arabic.	Carter.
? *Beri-beri* (bad sickness).	Singhalese.	Simon.

The Japanese word is *Kakke* = disease of the legs.
,, French ,, *Barbiers.*
,, Malay (Java) ,, *Lm'poli* = paralysed.

It has many other local names, and many Latin or Greek terms have been suggested by authors dissatisfied with the current nomenclature (*e.g.*, Neuritis multiplex endemica—*Scheube*).

History and Geographical Distribution.—An Arabian

campaign by the Romans, under Ælius Gallus in B.C. 24, was attacked by an epidemic (described by *Dio Cassius*), which seems from the description to have been due to beri-beri.

In a Chinese pamphlet of the 2nd century mention is made of Kakke, so that the definite recognition of the condition is of considerable antiquity.

Baelz and *Scheube* were the first to show that beri-beri is in the nature of a specific peripheral neuritis.

Beri-beri has a very wide tropical and sub-tropical distribution, and is occasionally seen in temperate climates in the form of imported sporadic cases, ship epidemics, or isolated epidemics in institutions.

It has, *par excellence*, three endemic centres:—

The *Malay Peninsula and Archipelago.*
The main island of *Japan.*
The coast and interior of *Brazil.*

In former years it was very prevalent in India and Ceylon, but is now less so, although small endemic centres persist, as on the Masulipatam Coast, &c.

Other centres are:—The Philippines (unknown before 1882); Burmah; Siam; Shanghai; Amoy; Swatow; Foochow; Hong Kong; parts of the West and East Coasts of Africa; Madagascar and Mauritius; amongst the Chinese in Australia; Fiji; Sandwich Islands; Central America; Cuba; and a few of the other West Indian Islands.

Cases are frequently imported by ships into the harbours of Europe, but the disease does not seem to spread.

In 1894 to 1898 several asylums in England and America reported cases of beri-beri, notably the Richmond Asylum, Dublin, where a sharp epidemic took place as follows:—

Year.	Lunatics.	Cases.	Case Mortality.
1894, . . .	1503	174	Per cent. 14·3
1895, . . .	...	...	...
1896, . . .	1686	114	7
1897, . . .	1800	246	4·4
1898, . . .	...	13	...

thus making a total of 547 cases in five years, of which 15 occurred amongst the attendants.

Etiology.—The specific cause and method of propagation of beri-beri have caused endless discussion, and are still shrouded in mystery.

Some of the many theories may be here put forward and briefly discussed:—

1. *Gelpke's Theory.*—That the disease is due to a dried fish infected by a trichina.

2. *Grimm's Theory.*—That it is caused by ingestion of infected fish.

3. *Miura's Theory.*—That it is caused by ingestion of certain kinds of raw fish.

Remarks.—These three theories can be excluded by the results of Hamilton Wright's experiments in the Pudoh Gaol, Kwala Lumpor. In 1901 for eight months no fish at all was included in the diet of the 49 prisoners, who, during that time, contracted the disease.

4. *Ross' Theory.*—That it is due to arsenical poisoning.

Remarks.—Eleven months continuous observation in the above gaol disproved the presence of arsenic in any shape or form. There was no disturbance of soil, no tinned food, and no contaminated sugar. Moreover, it would be absurd to imagine that the 90 cases which originated during those eleven months would not have manifested for two weeks to eighteen months the effects of arsenic absorbed previous to their imprisonment.

5. *Takaki's Theory.*—That it is caused by nitrogen starvation.

Remarks.—Considerable attention was called to this theory by the decrease in beri-beri amongst the men of the Japanese army and navy after alteration of the diet scale; but, as *Baelz* has pointed out, the decrease took place in all barracks, although the change was purposely not effected simultaneously. Wright's Kwala Lumpor experiments showed a diet of 1 : 12 :: N : C (normal = 1 : 15 :: N : C) which was supplied for eight months, during the latter half of which many cases occurred. This theory can therefore be excluded.

6. *Glögner's Theory.*—That it is due to a hæmamœba.

Remarks.—Careful search has been made in so many cases by numberless skilled observers, that, had such plasmodium been present, it would almost certainly have been found.

7. *Braddon's Theory.*—That it is caused by ingestion of a specific organism growing on mouldy rice.

Remarks.—In the above Kwala Lumpor experiments all the rice used in the gaol was cooked in iron steamers for two hours under a pressure of two atmospheres. Neither organism nor toxin should be able to withstand this treatment, and, therefore, whether it be a cause or not, beri-beri can be otherwise produced.

8. *Hose's Theory.*—That it is due to the ingestion of mouldy rice.

Remarks.—Rice is not eaten by natives when uncooked. The usual ɔoking should be sufficient to destroy all moulds—and the cooking in ɑaols and other public institutions, by steam, should likewise destroy any ɪngus toxins; nevertheless beri-beri continues.

Hose's experiments with three monkeys (*Macacus nemestrinus*) are ɪtirely inconclusive, since the cardinal symptoms of a peripheral ɜuritis with œdema were not produced, and all that resulted was a lack of energy" shown by two of them.

The feeding of fowls on bazaar rice resulting in "weakness" and paralytic symptoms" are also inconclusive, since *Maurer* produced the ɑme results by feeding fowls on oxalic acid.

That a rice diet cannot be considered to be the cause of the disease is ɪrther exemplified by the occurrence of beri-beri in non-rice eating ɔuntries (*Scheube*) such as Brazil, the Moluccas, &c., where sago, fish, ɪd game are the staple articles of diet (*Fiebig*), and where the disease ·tacked Europeans who had never eaten rice (*Voorthuis*).

9. *Manson's Theory.*—That it is due to a place germ—earth, ɔor, or house—which distils a volatile or stable toxin, of which the halation or ingestion causes the disease.

Remarks.—In no disease at present known, of which the specific ·igin is established, have we any similar method of infection by toxic ·halation. In all such cases the toxins are elaborated by the micro-ganism *within* the body of the human host; nor do we know of any ·ample of a toxin emanating from bacteria in earth, floors, &c., and ·fecting food—unless the actual bacteria or moulds themselves gain ·ccess to the foodstuff.

10. *Laurent's Theory.*—That it is caused by a deficiency of fat in ɪe diet.

Remarks.—The fat theory has been disproved by many observers.

11. *Trentlein's Theory.*—That it is due to oxalate poisoning.

Remarks.—*Maurer's* results in producing a beri-beric condition in ·wls by feeding them with oxalic acid, have been confirmed and ex-nded by *Trentlein*, who used oxalic acid, oxalates, and rice meal. he heart and peripheral nerves showed fatty and degenerative changes ·uch as in beri-beri. *Trentlein* attributes the condition to a removal calcium from the affected tissues by the action of oxalic acid or its ·lts, either administered as such or produced from the rice meal by e agency of micro-organisms present in the crop of the animal.

He arrested the symptoms and checked the pathological changes by ·ving an excess of calcium carbonate in the food, and so forming soluble calcium oxalate.

In five cases of acute wet beri-beri, he found a pathological excess calcium oxalate in the urine.

12. *Pekelharing and Winkler's Coccus.*—That it is due to a white ·quefying coccus, requiring repeated introduction.

Remarks.—In 80 cases examined, 65 were sterile; 6 showed white ·uefying cocci; and 9 cases various other bacteria.

In experimenting with these strains, 9 out of 12 animals gave ·sitive results with the white liquefying cocci. In these cases, however,

the degeneration of certain nerves was held to be a positive result, and all the other pathological and clinical changes were disregarded.

13. *Hamilton Wright's Bacillus.*—He considers that a certain specific bacillus lies dormant in certain localities; that it gains access to the body by the mouth, giving rise to a primary duodenal lesion. The resulting toxin produces the characteristic effects on the peripheral nerves, while the organism itself escapes in the fæces.

Remarks.—*Wright* maintains that nearly all cases fatal in the acute stage exhibit a neurosis of the gastro-duodenal mucosa: that a bacillus is constantly present: that in the acute stage the nerve changes are merely toxic effects, and only show degeneration in a stage of residual paralysis.

Dudgeon, who has investigated the bacillus, finds that it has no action on animals, and is not agglutinated by the sera of beri-beri patients.

14. *Tsuzuki's Coccus.*—*Tsuzuki* has isolated a diplococcus, not from the blood but from the urine—the *Micrococcus beri-bericus*—0·7 to 0·8 μ long by 0·4 to 0·5 μ wide; stains with gram; non-motile; ferments grape- and milk-sugar; no gas; small semi-transparent colonies on agar; clots milk after a week; resists a temperature of 60° C. for one hour.

Remarks.—Positive reaction was obtained by serum tests in 103 out of 106 patients examined, 26 controls being all negative.

The coccus was isolated in about 28 per cent. of the cases examined, 65 control urines giving negative results.

It was also isolated from the fæces of beri-beri patients in about 58 per cent. of cases.

Animal inoculations of the toxin resulted in cardiac and paralytic clinical forms, as in man; and the pathological changes were apparently identical.

From consideration of the above hypotheses, it is obvious that the definite cause of the disease still requires elucidation.

Theories 11 and 14, above, are sufficiently plausible to warrant the attention of tropical workers in endemic areas.

General Conclusions and Etiological Factors.—Although the specific cause has not yet been discovered, it is almost certainly an extra-corporeal animal or vegetable parasite, entering the body by one of the usual channels, and there producing the toxin which causes the characteristic nerve degeneration.

There is no evidence to incriminate any definite food, or drink, or intermediate host.

Its behaviour simulates that of malaria in a few particulars, such as:—

(*a*) Frequent occurrence after disturbance of the soil.

(*b*) Relapses occurring after long intervals, without likelihood of re-infection.

(*c*) Predilection for new-comers.

Race and nationality play an important part. The disease is very

:are amongst Europeans, less so amongst half-eastes, and most ommon amongst native raees.

The *male sex* is far more often attaeked than the female.

With regard to *age*, the greatest liability is from *15 to 30*. The oungest ease reported is by *Graham*, in Sumatra, in a Chinese boy f five. One of *Scheube's* patients had attained 65.

In Japan the disease is one ehiefly of the middle elasses (*Scheube*). On the other hand, in the Malayan Peninsula and Arehipelago it perhaps twenty times more eommon amongst the lower than mongst the middle elasses.

Predisposing causes would seem espeeially to eonsist of warm, amp atmospheres, overerowding, and indifferent foods. It is specially eommon, for instanee, in gaols, barraeks, asylums, mining oolie-lines, erews' quarters on shipboard, &e.

Daniels, who studied the subject for a short time, when director of 1e Research Institute at Kwala Lumpor, summarises his *conclusions* s follows :—

1. That beri-beri is an infeetious disease. As a rule, a short period f incubation and a period of exposure of less than three months is :quisite for full development of the disease where the endemic index high.

2. That there is no definite proof that an intermediate host is re-.iired, but the balance of evidence is against its being conveyed by irth, air, water, or food, or contamination with sewage or other ecal matter.

3. That there is some evidence that for a short period only after the ccupation of small spaces, beds, bedrooms, &c., the "poison" or irrier of infeetion may remain.

4. That food, either as regards quantity and quality, its nature or :lative proportions, may have an effeet on the susceptibility of the atients, though the proofs are not eonclusive, but is not the eausative gent.

5. That if an intermediate host for the unknown parasite is required, must be either a *cimex* or a *pediculus*. That pediculi as earriers ould better explain the ineidenee of the disease than any other blood-ıcker.

6. That a eloser enquiry into the earlier stages of the disease is quired. That where opportunities for such an enquiry occur, renewed tention should be bestowed on the blood and tissues, with a view to etermining the presence or absenee of any protozoon.

7. *Prophylaxis.*—That in view of the failures of various attempts at sinfection of buildings and places, and of various modifieations of et to have marked effects, more attention should be paid to limiting e ehances of personal infeetion, and that particular attention should paid to the personal eleanliness, freedom from vermin, and isolation early or trivial cases of the disease.

Table of Beri-beri Incidence, showing the general ineidenee in e town of Singapore for twenty years, as gauged by the admis-ons to the Pauper Hospital, eompared with the ineidenee in the ngapore Gaol for the same period :—

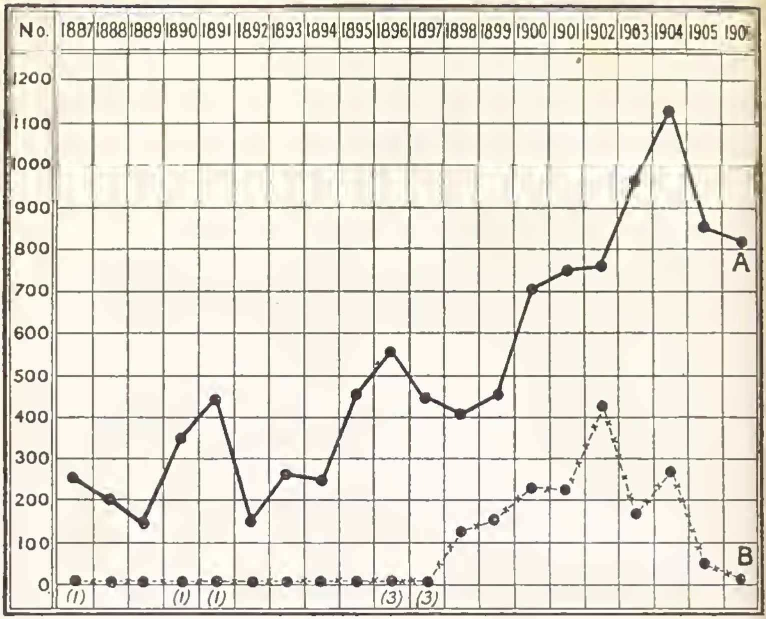

Fig. 29.—Table of Beri-beri Incidences.—**A**, Admissions to Pauper Hospital, Singapore; **B**, Cases occurring in Singapore Gaol.

Remarks.—This table is of interest as showing a certain similarity of curve, leading to the supposition that the incidence in a gaol bears a more or less direct relation to the number of cases introduced from outside.

Symptomatology.—**Classification.**—Many schemes of classification have been propounded by authors. Of these, *Scheube's* four clinical types are undoubtedly the best :—

1. Rudimentary.
2. Atrophic.
3. Œdematous.
4. Pernicious.

Incubation Period.—*Pekelhäring* and *Winkler* considered the incubation period to be a long one. *Hamilton Wright*, on the other hand, from a series of observations on Sinkhehs (or contract coolies) brought from China to the tin mines of the Federated Malay States, came to the conclusion that *10 to 30 days* is the normal incubation period; and this seems to coincide with subsequent observations, although he overlooks the possibility of infection whilst detained *en route* in the closely-locked and barred lodging-houses of Singapore.

A case under the care of the author is of interest as showing a robable incubation period, and early symptoms:—

A Chinese hospital attendant, aged 31, joined the Government rvice at the quarantine station of the port of Singapore on 1st Oct., 906. He was of exceptionally strong build, and in every way healthy. is quarters consisted of a private room in a barrack, similar in all spects to those occupied by the other six hospital attendants, all of hom were healthy and well. No cases of beri-beri existed at the arantine station either at the time or for many months previously.

On 25th Dec. he proceeded ashore to Singapore on twenty-four ours' leave, after which he returned.

On 5th Jan., 1907, he complained of weakness of arms and legs, me tightness of the chest, and eructations after food; there was also ight frontal headache. The bowels were open, and appetite good.

6th Jan.—Legs weaker, great difficulty in walking, headache persists.

7th Jan.—Cannot walk at all without help.

8th Jan.—Pulse 88. Temperature and respirations normal. Cannot and. Pretibial œdema. Knee-jerks absent. KSCN absent from liva. The appetite good and bowels open. Slight tenderness of calf uscles. The blood was carefully examined, and showed nothing normal. The food taken by him during his three months at the arantine station was from the same supply used by the other six althy attendants, and cooked in the same pots.

On 9th Jan. he was removed to his home in Singapore.

On 4th March he was seen again, and his condition was unaltered.

Notes.—It is very hard to explain this case unless we conclude that became in some way infected during the one day spent in Singapore. so, there must have been a definite incubation of ten days.

No beri-beri was known to exist in the place where he slept when hore, but he had two meals at eating stalls in different parts of the wn. He was not shaved when ashore. The tentative inference, erefore, is against infection by bed-bugs or lice, and rather in favour her of food or mosquitoes.

Symptoms.—As a rule, there is no prodromata. The first mptom usually noticed is a weakness of the legs. At the same ne a little epigastric fulness or discomfort is usually remarked on.

If seen at this stage, the patient can walk, but with difficulty. he calf muscles are slightly tender. The knee jerk is feeble and ually lost by the third day. There is slight œdema over the bia.

These symptoms gradually become aggravated; and, in bad cases, e patient can scarcely walk at all, even with help.

Having reached this stage, one of four things may happen.

(*a*) The case may continue as a rudimentary or mixed form.

(*b*) It may develop into an œdematous type (or wet beri-beri).

(*c*) It may become atrophic (dry beri-beri); or

(*d*) In any of the above three conditions (most often in the lematous variety) it may fulminate, and is then known as pernicious ri-beri.

(*a*) In the *Rudimentary cases* the initial symptoms become gravated. Numbness extends over the body. Palpitation and

epigastric oppression are complained of. There are often dyspeptic symptoms. The tibial œdema persists, and the calf muscles become very tender.

This condition may persist for a few weeks or even months.

At any time during its course, the disease may become fulminating, but this is unusual in this type.

As a rule, there will be a gradual improvement until convalescence is established.

Relapses or re-infections, however, are extremely frequent.

The death-rate is quite low.

(*b*) In the *Atrophic form* the initial symptoms progress, but the speedy advance of the paralytic features is most noticeable; as a rule, it is confined to limbs and trunk, but it may affect the face, tongue, pharynx and larynx.

The œdema and cardiac symptoms are not marked.

The hyperæsthesia is generally a painful feature.

The affected limbs become wasted to the smallest proportions—almost skin and bone.

Intercurrent disease frequently ends the scene. If it does not, recovery is extremely slow, and months and years may elapse before it is partially complete.

(*c*) In the *Œdematous type* the initial symptoms become differentiated by the accentuation of the cardiac and œdematous features.

The œdema gradually spreads all over the body, and is accompanied by effusions into the serous cavities, palpitation and dyspnœa.

The secretion of urine is diminished and analysis will show an excess of oxalates, and an absence of albumen.

In bad cases the patient becomes enormously swollen, the whole body being like an inflated bladder. The paralysis is not generally so marked as in the atrophic form, but there is great tenderness on pressure over the affected nerve trunks.

In this stage the disease may remain for weeks, with alternately increasing and diminishing symptoms.

This œdemic form of beri-beri is the one in which pernicious symptoms most usually occur.

In these, (*d*) *fulminating attacks* the dyspnœa becomes more urgent, so that the patient has to sit up in bed and struggle for breath. There is intense pain in the region of the sternum and epigastrium. Cardiac failure and cyanosis quickly supervene. The pulse is rapid, intermittent, and small. Vomiting may or may not occur. There is usually aphonia; and death—an agonising death—from an overdistended right heart and pulmonary œdema soon ends the scene.

Occasionally in this, as in other types of beri-beri, death may occur suddenly from syncope.

If the cases do not fulminate, convalescence may be gradually established. Ataxic cases may slowly improve and begin to walk; œdematous cases may resolve, with profuse diuresis and diaphoresis,

[*To face p.* 140.

Fig. 30.—Beri-Beri. *Showing wasting of the gastrocnemii and peroneal muscles and drop-foot action in walking.* [Photo. by Dr. Dane, Singapore.]

[To face page 141.]

DIFFERENTIAL DIAGNOSIS.

	Beri-beri.	Alcoholic Neuritis.	Laudry's Paralysis.	Locomotor Ataxia.	Spastic Paraplegia.	Lumbar Myelitis.	Pellagra.	Ergotism.	Lathyrism.
History, . . .	Practically nothing to be made out except an occasional previous attack.	Frequent imbibings. —— Neuralgias. —— Muscular tenderness.	Acute diseases. —— Cold. —— Alcohol. —— Syphilis.	Syphilis. —— Lightning pains. —— Argyll-Robertson Pupil.	Slow onset with asthenia and stiffness.	Rapid onset after—Injury, smallpox, syphilis, &c.	Eating maize. —— Winter relapse.	Eating rye. —— Convulsions. —— Gangrene.	Sudden onset. —— Eating pulse.
Knee jerks, . .	–	–	–	–	+ +	–	+	–	+ +
Urine, . . .	Chlorides – Oxalates + +	Albumen.	Normal.	± Incontinence.	Normal.	Incontinence.	± Albumen.	Diminished.	± {Retention or Incontinence.
Œdema, . . .	Always tibial. Sometimes general.	Probable.	Nil.	± Feet.	Nil.	Nil.	Late.	Nil.	Nil.
Sensation, . . .	Impaired or lost.	Lost or hyperæsthesia.	Rarely lost.	Plantar anæsthesia	Numbness.	Crural anæsthesia.	Lost.	Lost.	Normal.
Trophic changes, .	Muscular Wasting.	Atrophy of affected muscles.	Nil.	± Perforating ulcer of foot.	Nil.	Atrophy.	Marasmus.	±	Nil.
Tenderness, . .	Of calf muscles. Extreme.	+	±	Nil.	Nil.	Nil.	Nil.	Nil.	Nil.
Ankle clonus, . .	–	–	–	Nil.	+	–	–	–	+
Sex, ...	More often in males.	More often in women.	Usually males.	Generally in males.	Often in males.	Generally in males.	Males = females.	Males = females.	More often males.
Age, ...	18-30	Middle life.	20-30.	30-40	Early adult life.	Adult.	40-60	Middle life.	Youth.
Pathology, . .	Degeneration of sheath of Schutner and of axis cylinders.	Extensors most involved.	Often no lesions found.	Degeneration of posterior roots and dorsal columns.	Sclerosis of crossed pyramidal tract.	Progressive softening of cord.	Sclerosis of posterior and postero-lateral columns.	Degeneration of posterior columns.	Slow sclerosis of lateral columns.
Gait, ...	Foot raised high. Ankle drops. Toes first on ground.	Toes raised. Foot slapped flat like a flail.	Complete paralysis in a few hours.	Leg thrown violently. Foot raised high. Heel first to ground.	Legs moved stiffly. Toes drag on the ground.	Complete paraplegia.	Paralytic.	Not pathognomonic.	Legs rigidly extended. Feet rotated inwards. Toes flexed.
Course, . . .	Acute, chronic, or relapsing.	Recovery after many months.	Begins legs, extends to trunk and respiratory muscles. Usually fatal in few weeks.	Frequently lasts for years. No recovery probable.	Very chronic.	Cystitis and fæcal incontinence.	Marasmus and intercurrent disease.	Chronic, with gangrene.	Mortality low. Recovery rare.

Pellagra.	Ergotism.	Lathyrism.
maizc. ——— relapse.	Eating rye. ——— Convulsions. ——— Gangrene.	Sudden onset. ——— Eating pulse.
+	–	+ +
umen.	Diminished.	± { Retention or Incontinence.
	Nil.	Nil.
	Lost.	Normal.
mus.	±	Nil.
	Nil.	Nil.
–	–	+
= females.	Males = females.	More often males.
	Middle life.	Youth.
sis of posterior d postero-lateral umns.	Degeneration of posterior columns.	Slow sclerosis of lateral columns.
tic.	Not pathognomonic.	Legs rigidly extended. Feet rotated inwards. Toes flexed.
mus and inter- rent disease.	Chronic, with gangrene.	Mortality low. Recovery rare.

l gradually assume an ataxic type during the long road to con-
escence. The cardiac dilation will adjust itself and the bruits
appear.

On the other hand, a case may appear to have quite recovered and
n will relapse. . . .

Diagnosis.—The important points are :—

(*a*) Serous effusions.
(*b*) Pre-tibial œdema.
(*c*) Tenderness of calf muscles.
(*d*) Paralysis and wasting in legs and thighs.
(*e*) Loss of patella reflex from second or third day.
(*f*) R. ventricle enlarged. Second pulmonary sound accentuated.
cond mitral sound reduplicated.
(*g*) Slightest exertion increases pulse twenty to thirty beats per
nute.
(*h*) Characteristic gait.

Differential Diagnosis.—The following conditions have some
nts in common with, or different from, beri-beri, which it is well
bear in mind :—

Alcoholic neuritis; Laudry's paralysis; tabes; spastic paraplegia;
ibar myelitis; pellagra; ergotism; lathyrism.

The subjoined table will show the chief details of the differential
gnosis.

Treatment.—If possible, the patient should be removed from
building or locality in which the disease was contracted. If, as
infrequently happens, the disease is contracted in one place and
exhibited in another, then the need for removal is, of course,
viated.

The food should be nutritious and easily digested, and should
ntain a sufficiency of nitrogen and fat.

A good routine prescription is the following :—

℞.—Mag. sulph., ... gr. lx.
Ac. hydrochlor. dil., ... ♏xx.
Tct. aurantii, ... ʒi.
Inf. calumbæ ad . . . . . ℥i.

M. F. Mist.

Sig.—℥i. t. d. s. Continue for a week, and repeat after a few days' intermission.

If there is much œdema the following mixture is often of value :—

℞.—Liq. ammon. acet., ... ʒi.
Pot. nit., ... gr. x.
Pot. acet., ... gr. xv.
Aq. camph. ad . . . . . . ℥i.

M. F. Mist.

Sig.—℥i. t. d. s.

If the heart shows signs of failure, give—

℞.—Tct. digit., ...	♏x.
Ammon. carb., ...	gr. x.
Sp. œth. co., ...	ʒi.
Aq. ad	℥i.

M. F. Mist.

Sig.—℥i. every four hours.

Beyond this, but little can be done in the way of routine treatment. The symptoms should be treated as they arise.

For the muscular atrophy faradisation and massage should be tried as soon as the hyperæsthesia has subsided. Sea voyages are often valuable aids to a cure.

For the paresis *Hewlett* advocates strychnine, gr. $\frac{1}{60}$, injected into each thigh daily, but *Scheube* purports to have found this treatment useless. In all severe cases rest in bed is essential.

Morbid Anatomy and Pathology.

Rigor mortis is short. A post-mortem rise of temperature is sometimes seen. The author has found a temperature of 104° F. in the thoracic and abdominal cavities three hours after death.

The *heart* is usually enlarged and the right ventricle dilated.

The *myocardium* is fatty.

The *blood* is dark-red and very fluid.

The *lungs* are generally œdematous.

There is often some hyperæmia of the *intestinal mucosa*, especially in the stomach.

The *liver* is usually congested, and the spleen generally enlarged.

The *kidneys* are often engorged, but nephritis is rare.

The most important changes are those of the *nervous system*.

The *peripheral nerves* are the principal seat of the disease. Microscopically they show inflammatory degeneration.

The myelin sheath breaks up into beads, and finally disappears; and the outline of the axis cylinders become indefinite, until the whole disappears.

In chronic cases the endoneurial and perineurial nuclei are increased, and, finally, connective tissue replaces the nerve.

The nerve trunks suffer but little by these changes, which are chiefly confined to the muscular branches.

In acute cases the vagus and its branches become involved.

Ellis found that the pathological changes included the participation of the nerves to the arteries, the splanchnics, and the solar and renal flexuses.

In addition to the nerve affection, the *muscles*, especially those of the calves, show fatty and colloid degeneration.

The spinal cord is never primarily diseased in beri-beri, although occasionally it may be implicated *via* the nerve roots.

The serous exudation of the œdematous type is due to the implication of the vaso-motor nerves.

In the atrophic type the nerves affected are chiefly the motor and sensory muscular branches.

The occurrence of a pernicious attack implies the involvement of the cardiac vagi.

The diminished urinary secretion depends partly on the implication of the renal nerves, and partly on the low blood pressure.

The brain changes are usually slight, and consist of a little hyperæmia or œdema of the meninges or brain substance.

CHAPTER XIV.

BILHARZIOSIS.

Definition.—An infection of human beings by a trematode, the *Schistosomum hæmatobium* (Bilharz, 1852), characterised by hæmaturia and by the presence of the ova in the urine, fæces, or both.

Synonyms. — I. *Of the parasite:* — *Bilharzia hæmatobia*, Cobbold; *Distoma hæmatobium*, Bilharz, 1851; *Distoma capense*, Harley, 1864.

II. *Of the disease:—Bilharzia disease; Endemic hæmaturia.*

History and Geographical Distribution.—The parasite was discovered in 1851 by *Bilharz* in Cairo.

Probably the hæmaturia referred to by *Renault* during the French occupation of Egypt in 1878 was the same disease.

In honour of its discoverer the parasite was called "Bilharzia hæmatobia" by *Cobbold.*

Harley in 1864 found the ova in cases of hæmaturia in Natal.

Its range of endemic distribution extends over a large part of Africa:—East, West and Central Africa; Tunis; Algiers; the Cape; Mauritius; Syria; Cyprus; Mecca. Cases have also been reported from Penang, Shanghai, Illinois, and probably the West Indies.

Natural History.—The species *Schistosomum hæmatobium* (Fig. 31*a*) belongs to the genus *Schistosomum;* the family *Schistosomidæ;* order *Malacocotylea;* class *Trematoda.*

The *male* is whitish. It becomes mature when 4 mm. long, but subsequently attains a length of 12 to 14 mm. There are two sucking discs on the front of the body, oral and ventral, placed close together. Behind the ventral sucker the body broadens to a width of 1 mm. while decreasing in thickness. The lateral edges curl in ventrally, forming an almost closed canal—the gynœcophoric canal—in which the female can repose.

On the dorsal surface of the posterior part of the body are spinous papillæ used to grasp the vein walls.

The excretory pore is somewhat dorsal, and is at the posterior end.

The genital pore is ventral and anterior, being situated behind the ventral sucker.

The *females* are filiform and pointed at each end. In length they reach about 20 mm., and in breadth 0·2 mm. As in the male, there is an oral and a ventral sucker. The genital pore lies immediately behind the ventral sucker. For purposes of copulation, the female

lies in the gynœcophoric canal of the male, the two genital pores being approximated. As there is no penis, the semen is discharged into the canal, and is probably taken up by imbibition (*Sonsino*).

The *eggs* are oval, yellowish and slightly transparent; enclosed in a thin shell with a spine—usually terminal, sometimes lateral. They vary in size, but are generally ·16 mm. by 0·06 mm. (Fig. 32.) These eggs contain a ciliated embryo, termed a *miracidium*.

If the eggs remain in the urine or fæces they, as well as the enclosed embryo, will perish.

If, however, water is added, the embryo exhibits violent movements and very soon ruptures its containing shell, and swims about contentedly in the liquid.

Manifestly, therefore, the first extra corporeal life-phase of the embryo must be passed in water. What the subsequent development may be is as yet unknown. Analogy would lead us to suppose that some intermediary host exists (as in the case of "cyclops" and the guinea-worm embryos), and that subsequent development may take place in the body of this unknown fresh-water creature, and thus in turn reach a fresh human host.

Or, again, it is quite possible that the miracidia which have gained access to the pools may be able to enter the skin of natives or others standing in the water, in the same way that the larvæ of ankylostomes have been proved to do.

The life of the adult worm is probably a long one. *Sonsino* mentions the case of a boy who suffered from bilharziosis when leaving Egypt, and subsequently lived in France for nine years, during the whole of which time living ova continued to be evacuated.

Etiology.—The male sex seems more liable to attack than the female; country folk than those of the towns; natives than Europeans. The native habit of frequent bathing and washing in rivers, pools, &c., might account for this partiality; and, if so, would seem to point rather to the skin as the pathological channel of entrance.

Griesinger's observations in Egypt seem to show the greatest incidence to occur during the months of June to August, which approximately corresponds with the rise of the Nile.

The *incubation period* appears to be variable.

It is given as four weeks (*Hatch*) to four months (*Brock*).

Pathological Anatomy.—The *habitat of this adult trematode* the *portal vein* with its tributary intestinal branches, especially those of the rectum and bladder.

The veins of the bladder are connected with the rectal veins through the prostatic plexus.

These rectal veins discharge partly into the portal veins *via* the superior hæmorrhoidal veins; and partly into the inferior vena cava *via* the middle and inferior hæmorrhoidal veins.

At puberty the worms pair, the female lying in the gynœcophoric canal of the male. Locomotion in the venous system is brought

about by the males, who, with the females, descend towards the bladder and rectum to deposit their eggs—evidently with the idea of giving them the best chance of reaching the element necessary for their further development.

The number of adult worms found in one person is often large (*Kartulis* reports 300).

The small veins and capillaries are torn or bored through by the numerous ova which block them up, and thus get free into the tissues and form the nucleus of the inflammatory changes known as *Bilharzial infarction.*

The following are the chief pathological conditions found in the various organs:—

The Bladder.—This is the commonest site for invasion by ova. The walls are usually thickened by muscular hypertrophy. The mucous membrane shows reddened and swollen patches exuding blood-stained mucus with numerous eggs. Excrescences, nodules, and raised hard lamellæ are found scattered about, especially in the trigone of the bladder.

Calculi are very frequently found, both in bladder, ureters, and kidneys. They consist, as a rule, of oxalate of lime, and are probably due to the infection, since ova have been frequently found in the calcular nucleus.

Under the microscope it is seen that the submucosa of the bladder is much hypertrophied. There is a large leucocytic infiltration; and an immense deposit of ova, some of which become calcified.

The *vesiculæ seminales* are frequently affected, with thickened and infiltrated walls containing many calcified ova.

The *prostate* is usually much enlarged and hardened, owing to the deposit of ova.

The *rectum* frequently shows changes similar to those in the bladder. The deposit of ova is greatest in the submucosa.

Hypertrophied glandular layers will form polypoid excrescences (often mistaken for piles) in which many ova are to be found, and sometimes parent worms.

In the *vagina,* bilharziosis may lead to a sub-acute vaginitis, and a thickening of the mucous membrane of the posterior wall having well-marked crossing fissures on its surface (*Milton*).

Urinary fistulæ are not uncommon, and are usually secondary to bladder infection. They are of two kinds—roof fistulæ and floor fistulæ.

In the case of roof fistulæ, as a rule, the condition passes almost unnoticed till a perineal subcutaneous abscess is formed. These give rise to one or more sinuses in the perineum, scrotum, pubes, or thighs, but away from the direct line of the urethra. The openings are not large, and some granulations may protrude.

In the case of floor fistulæ, a periurethral abscess is formed, with a subsequent inflammatory stricture. Sinuses will be in the urethral line, either in the perineum or penis. There is greater induration

Fig. 31*a*. Female (*S. hæmatobium*) in gynæcophoric canal of male.

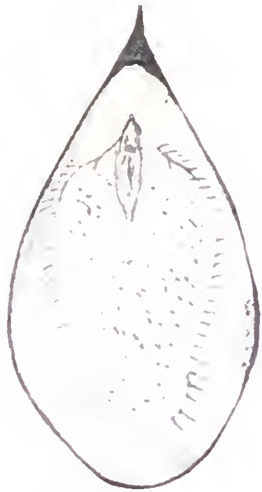

Fig. 32. Ovum of *Schistosomum hæmatobium* (× 500).

Fig. 31*b*. Ova.

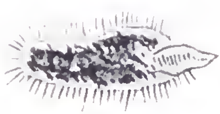

Fig. 31*c*. Ciliated miracidium, escaped from ovum.

in the neighbourhood: pus oozes from the urethra, and the meatus is frequently narrowed by scars.

Bilharzia ova have also been found in the following organs and places:—*Mesenteric glands*; *lungs*; *liver*; the *cutis*; a *gall stone*; and in the *blood* of the left ventricle.

Hitherto search in the spleen and pancreas has been without result.

Symptoms.—The symptoms will vary very much. In many cases no trouble may occur at all.

The principal symptom is *hæmaturia*. There is frequent micurition, with very little pain; but a slight irritation may be felt when passing urine.

The last few drops which are passed will be found to be blood-tained, and if this is microscopically examined, it will show ova, pithelium, blood cells, and perhaps a deposit of urinary crystals. A low-power objective is quite sufficient for diagnostic purposes.

The hæmaturia is occasionally increased by violent exercise, or gastronomic excesses.

This condition of hæmaturia may last for months or years; and, lthough no re-infection may take place, the ova will still be passed or many years. In such favourable cases the hæmaturia will tend o decrease, and eventually the condition may be arrested when all he adult worms are dead.

Very often, however, the early slight symptoms may become xaggerated. Burning pains may radiate in the lower abdominal nd lumbar regions; the urine becomes alkaline, opaque, and of uniformly sanguineous colour; cystitis may supervene, with peraps paroxysms of pain from the passage of blood clots through the reters.

Urinary calculi in some instances form with calcified ova as nuclei.

Urethral fistulæ are found in 20 to 30 per cent. of the cases. hese fistulæ may be either in the roof or on the floor of the rethra. Roof fistulæ will lead to sinuses presenting usually in the erineum, away from the middle urethral line.

Floor-fistulæ, on the other hand, give rise to greater perirethral inflammation and pain, and present exteriorly in the middle ne, either in penis, scrotum, or perineum. They are often the use of stricture.

If the vesiculæ seminales are affected, spermatorrhœa may be set); and ova and blood will be found mixed with the semen.

Strangury and additional bladder trouble will follow the invasion the prostate.

If the rectum is involved, disorders somewhat resembling hæmoroids will be observed in the first instance. Some loss of blood, pecially at the end of defæcation, accompanied by itching and scomfort.

On digital examination many small soft excrescences will be und. Later, a regular dysenteric condition will be set up, with rmina and tenesmus as well as the passage of blood.

The vagina is not infrequently affected, a sub-acute vaginitis being set up, leading to a considerable thickening of the posterior wall.

If the liver and lungs are invaded by the ova, no symptoms usually result.

As a consequence of the frequent hæmorrhages and local inflammations, a condition of anæmia and emaciation frequently ensue, and death happens either from

1. Exhaustion.
2. Pyæmia.
3. Uræmia.
4. Intercurrent disease.

Diagnosis and Prognosis.—The passage of blood by the urethra or rectum occurring in persons in or from an endemic area should always arouse suspicion. Microscopical examination will clear up the diagnosis in such cases.

Bilharzial excrescences occurring outside the anus should not be mistaken for condylomata, nor the hard, round, rough lamellæ on the bladder wall for calculi.

Although the condition often causes but few symptoms and little inconvenience, yet it should always be regarded as serious, since death may so often occur as a result of its ultimate circumstances.

Life insurance companies will not accept patients (*Brock*).

In places where the disease is not extremely widespread, or in cases in which a patient has left the endemic area altogether, are naturally more favourable to the chances of a patient than if he were, for instance, in Egypt, since the event of re-infection is minimised.

Treatment.—This is at present somewhat unsatisfactory, since we have at present no means of eradicating the cause—viz., the adult worms in the portal system.

All we can do, therefore, is to mitigate the severity of the symptoms or rectify the morbid tissue conditions brought about by the deposited ova.

The following is a brief outline of the treatment adopted by Milton at the Kasr-el-Ainy Hospital in Cairo :—

For the *early hæmaturia* astringent bladder injections are not used, since they can neither cure nor relieve the condition. On the other hand, mild antiseptics, such as boracic acid, &c., will often afford some relief to the patient. At the same time, the internal administration of male fern is given in doses of 1 gramme of the liquid extract three times daily. This often acts like a charm in controlling and abolishing the hæmorrhage in a day or two. The rapidity of its action is against any effect on the adult worm, and it probably acts as a direct palliative on the mucous membrane of the bladder.

Cystitis should be treated by washing out the bladder, and by the internal administration of salol.

Calculi should be promptly crushed. Bad cystitis is no bar to

the performance of this operation, nor is it an indication for a lithotomy rather than lithrotrity unless the kidneys are the seat of bad secondary disease.

Disorganised bladders in the later stages are most difficult to deal with. The low vitality of the massive growth is often accountable for a sloughing necrosis after the mechanical injury caused by the insertion of a drainage tube.

If drainage is done at all it should be perineal, and not suprapubic. A "*Cock's* puncture" is the best method, followed by the introduction of two moderate-sized drainage tubes to facilitate washing out. These tubes should be retained for eight or ten days, after which the track will remain sufficiently patent to permit the passage of urine and sloughs. The patient's strength should meanwhile be kept up by constant attention to nourishment.

Urinary fistulæ should be treated by free and wide excision of the fistula and surrounding tissues.

Stricture should be dealt with by substituting a direct and fairly healthy escape for the urine through the perineum in place of the usual number of unhealthy tortuous fistulæ.

Vaginal invasion is best treated by excision of the thickened and infiltrated mucous membrane, provided the disease is sufficiently limited in extent.

In the *rectum* early irritation should be allayed by sedative and astringent applications.

Polypous excrescences are hard to deal with, as the disease generally extends too high up to be treated properly. The best treatment is to stretch or incise the sphincter, and swab the gut out as high as possible with a 1 in 10 solution of zinc chloride, applied on cotton wool by means of long forceps, and allowed to remain in contact for about a minute. It is then dried and flushed out with a copious saline enema.

Prolapse, if due to *Bilharzia*, and seen early in the disease, may best be treated by excision.

CHAPTER XV.

BLACKWATER FEVER.

Definition.—A tropical fever of doubtful origin, characterised by the presence of free hæmoglobin in the urine.

Synonyms.—*Hæmoglobinuric fever; bilious remittent fever; West African fever; yellow remittent; black jaundice; bilious fever, &c.*

History and Geographical Distribution.—The disease was reported on the West Coast of Africa by the French Colonial Medical Officers during the first quarter of the nineteenth century. In 1847, however, *Bryson*, in reporting on the diseases of that region, makes no mention of it.

In recent times, however, special attention has been given, and reports of the disease are forthcoming from various tropical countries. It is prevalent on the Gold Coast and in the Gaboon district; on the Congo; in Central Africa, and, to a less extent, on the East Coast. In Madagascar and Mauritius the disease has been not infrequently noticed.

Masterman reports it as often present in S. Palestine. Sporadic cases have been seen in India, Assam, and Cochin China by *Notter*, *Firth*, *Wenyon*, and others; also in Java, in Atjeh, and in New Guinea. *Freer* has treated a case at Penang (in the Straits Settlements). The Southern States, the West Indies, Central America, Venezuela, Guiana, Brazil, and Uruguay are all districts in which cases are found; and, coming nearer home, it has been observed in Spain, Italy, Sicily, Sardinia, Greece, &c.

Etiology.—This is very obscure, and the specific factor has not yet been discovered. Seasonal influences have seemingly nothing to do with the production of the disease, nor have those of sex. The disease is met with in both natives and Europeans, though the natives *who have lived for long in an endemic area* are less liable to attack than are Europeans.

Theories of Origin.—These are three:—

1. *That it is of malarial origin*, and due to some malignant, known or unknown, form of malarial parasite. This is the view which has perhaps most adherents amongst medical authors, and is supported by *Plehn*, *Scheube*, and others.

2. *That it is due to quinine poisoning.*—This view, also, has a certain number of adherents. *Koch*, *Stephens*, *Christophers*, and others are inclined to consider this the chief factor.

3. *That it is due to some specific cause of its own.*—*Yersin* has discovered a bacillus from two cases in Madagascar which he cultivated, and which had a toxic effect on rabbits and mice.

Smith and *Kilborne*, in 1889, showed the "Red-water or Texas fever" of cattle to be due to the presence of intracorpuscular parasites. These are called *Pyrosoma bigeminum*: and *Sambon* suggests that possibly an allied parasite may be the specific cause of blackwater fever.

In reviewing these theories and the arguments quoted to support them, it would certainly seem as if the third, that of distinctive specific origin, had most to be said in its favour.

1. *The Malarial Theory.—Scheube* says—"*It occurs in the most notoriously malarial regions*"; but so do many other diseases, including kala-azar, which Scheube places in his chapter on malaria, but which we now know to be due to *Leishmania donovani*.

Again *Scheube* says—"*Skilled observers, such as Plehn, Powell, &c., have found the small unpigmented seal-shaped malarial parasites peculiar to the tropical forms of malaria in the blood of blackwater fever patients, in most, if not in every case.*" In answer to this one can only say that (as Scheube himself states) *Rogers* found malarial parasites in all stages of kala-azar, and *Brown* "almost always found large and small malaria parasites, and also crescents" in that disease.

Moreover, we have another example of the danger of too hasty conclusions. In sleeping sickness the first cases examined showed *Filaria perstans*. Soon after the disease appeared in Uganda, *F. perstans* was found in the blood of nearly every case and was thought at the time to have a causal connection with the disease. But the weak point came in when it was shown that *F. perstans* occurred in regions where there was no sleeping sickness; that cases of sleeping sickness occurred without the presence of the filaria; and that in infected districts a large proportion of the general population also showed filarial infection.

In exactly the same way the malarial parasite is to be found in a thousand regions where blackwater fever is unknown; blackwater fever occurs in many recorded instances where malarial parasites are not found (see *Yersin's* cases, *Koch's* 23 out of 41 cases, *Plehn's* 11 out of 32 cases), and when, moreover, the non-parasitic malarial tests (*i.e.*, increased and pigmented large mononuclear leucocytes) are not present (see *Stephens* and *Christophers*, 6·3 per cent. of their Brit. Central Africa cases); and, lastly, that if the blood of the general population be examined, many may be found to harbour the malarial parasite.

Stephens and *Christophers* say—"*In a series of cases examined by ourselves in British Central Africa we found malaria parasites only in 12·5 per cent., but, as we have already shown, we have two further tests for a malarial infection:—*

"(1) *The increase in the percentage of large mononuclear leucocytes.*

"(2) *The presence of pigmented large mononuclear leucocytes.*

"*By using these tests we were able to prove that 93·7 per cent., not 12·5 per cent., of our cases were due to a malarial infection.*"

These are, perhaps, the least sound of any arguments. First, the increase in the percentage of large mononuclear elements is just as much pathognomonic of trypanosomiasis as it is of malaria, therefore why not say that such cases are due to trypanosomiasis; in either case neither the plasmodium nor the trypanosome are found.

Secondly, when conducting observations in a region so notoriously malarial as British Central Africa, to say that, in cases of blackwater

fever, the presence of pigmented large mononuclears is a proof of the malarial origin of the disease, is too illogical for consideration.

2. *The Quinine Theory.*—Of 43 cases of blackwater fever in Cameroon, F. Plehn noted that "*24 positively broke out a few hours after the administration of quinine; and of 55 cases treated by A. Plehn in the same place, 48 of the attacks were directly caused by quinine.*" "For this reason *Koch* has made the assertion that blackwater fever, as a rule, is solely quinine poisoning without malaria taking any part in the condition. *Baccelli* made the same assertion in regard to malaria hæmoglobinuria in Italy." *Ketchen* has reported a case from Capetown in which a hospital patient was noted to have an attack of hæmoglobinuria after each of several administrations of quinine, even after as small a dose as 1½ grains.

Stephen and *Christophers* also say—"*Blackwater fever is then a quinine intoxication, but it is something more. It occurs only in those who have previously suffered from malaria.*'

The remark as to malaria is pretty obvious. It is naturally (at the present day) highly unusual for people to be taking quinine in doses of any size unless it is for malaria. Therefore, if quinine is going to have a toxic effect, the presumption, in nine cases out of ten, is that there is a malarial condition as well.

But the whole case does not seem to be properly proved. Granted that in a few cases quinine might have such a toxic effect, is it likely that such cases of idiosyncrasy would be so few and far between amongst the thousands who are taking quinine the wide-world over, often in very large doses?

As *Osler* says—"So far as I have been able to judge from the recent records on the subject, the evidence on which the statement is based is very far from sufficient. Personally, too, I can say that using quinine in very large doses, as I have often done, I have never met with an instance in which it caused hæmaturia. On the other hand, in instances of acute albuminuria and of acute hæmaturia in malaria, I have seen the prompt and beneficial action of the drug."

So far as we can see, then, the case for malaria and the case for quinine, while indeed possible, seem to be highly improbable; and that the case should be much more scientifically strong to meet with general acceptance.

3. *A specific cause of its own.*—The analogies between this disease and Texas fever of cattle are most striking. For instance, where the disease is endemic it is notorious that the local native cattle become more or less immune. So with blackwater fever, in the endemic areas, the natives are much less susceptible than the new-coming Europeans.

The symptoms of Texas fever are—High fever, prostration, emaciation, hæmoglobinuria, anæmia of all organs with an icteric tint, enlargement of the spleen, &c. It is noteworthy that every one of the above symptoms is a counterpart of those of the human disease—blackwater fever.

Analogy would therefore lead us to suppose that a blood parasite somewhat similar to the *Pyrosoma bigeminum*, as yet undiscovered, might be the causative agent of the human disease.

In a thousand cases of Texas fever hæmoglobinuria will be practically constant; in a thousand cases of malarial fever the occurrence of hæmoglobinuria will be almost certainly nil.

If, therefore, one type of parasite gives rise to such constant and

definite clinical phenomena, the inference is that the same phenomenon in human beings will be caused by a definite specific and analogous parasite.

Symptoms.—Some slight malaise may precede the attack, but prodromata are rare. A rigor ushers in a febrile condition. The temperature (Figs. 33 and 34) rises fairly rapidly. It is nearly always accompanied by headache, general muscular pains, and vomiting. For two, or sometimes three, days the temperature continues to rise, falling somewhat in the morning and rising higher each evening. The vomiting continues and there is great weakness, with a fast low-tension pulse.

It is usually, however (and this is a point to which writers have

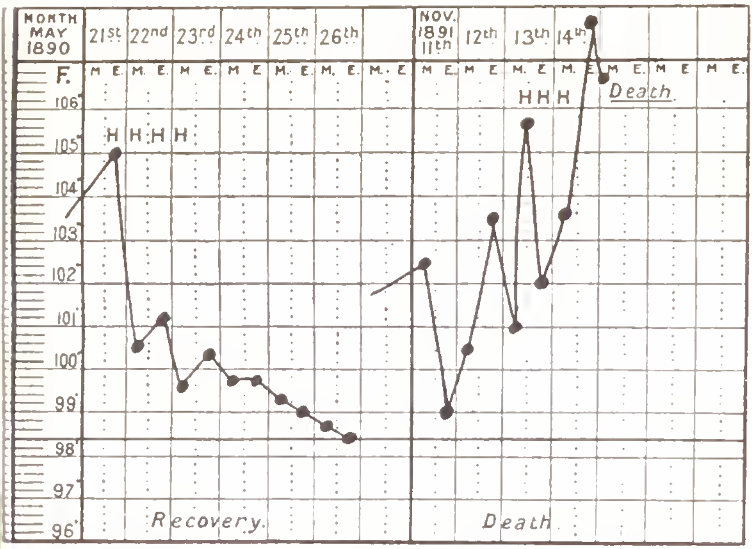

Fig. 33.—Two attacks of blackwater fever in the same person—1890 in London; 1891 on the Congo. H indicates hæmoglobinuria.

not as yet drawn attention), not until the second or third day, *when the temperature has reached its maximum*, that hæmoglobinuria is first seen. The urine is scanty, thick, and of high specific gravity. Examined microscopically it is found to contain renal and vesical epithelium, flakes of hæmoglobin, and hæmoglobinuric cylindrical casts. It is quite exceptional to find blood-corpuscles.

Very soon after the onset of the hæmoglobinuria a more or less intense general jaundice becomes established—probably not so much due to bile as to disintegration products of hæmoglobin.

The liver becomes enlarged and the spleen even more so, and there is frequently pain over both regions.

Retching and vomiting continue, and a persistent vomiting of green bile is almost invariably present in cases tending to a fatal termination. After a day or so of such symptoms the temperature generally falls, diaphoresis occurs, and the symptoms abate, the urine becoming again normal.

In fatal cases the temperature may remain normal or subnormal, the weakness becoming more marked; and suppression of urine occurs, death taking place from asphyxia, coma, or syncope. In other fatal cases there is a second or third rise of temperature with or without further hæmoglobinuria, until death occurs from coma, convulsions, or collapse.

In favourable cases the diaphoresis and fall of temperature will

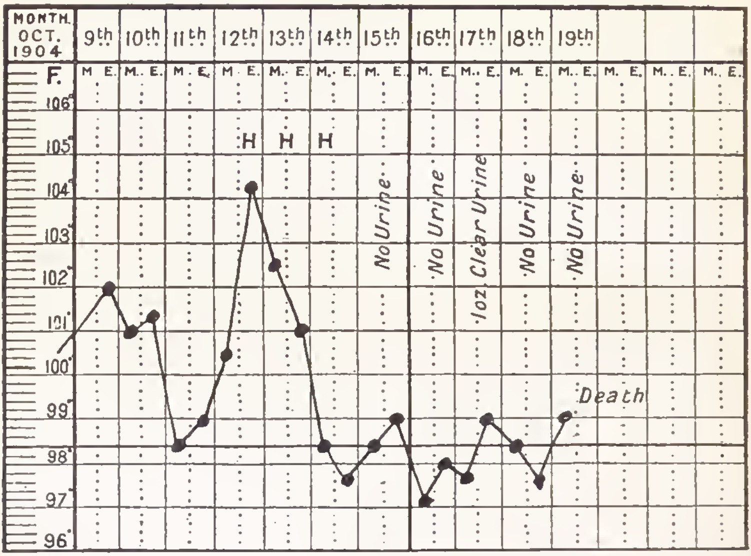

Fig. 34.—Fatal case of blackwater fever at Penang.

usher in a progressive convalescence, but with a very persistent albuminuria.

If the *blood* be examined it will be found to be thin and oily. The anæmia throughout is very marked.

The *case mortality* varies from 10 to 50 per cent.

Diagnosis.—Probably yellow fever is the only disease with which a mistake is likely to be made.

Ritchie considers that most, if not all, of the fatalities in new-comers to West Africa are due to blackwater fever; and also is of opinion that many of the so-called cases of blackwater fever are very probably yellow fever. The points of difference are :—

	Yellow Fever.	Blackwater Fever.
Vomit, . .	Black.	Green bile.
Urine, . .	Scanty. Albuminous. Occasional bile. Sometimes blood.	Scanty. Albuminous. Hæmoglobin.
Jaundice, . .	Present.	Present.
Spleen, . .	Normal.	Enlarged.
Stomach, . .	Injected.	Anæmic.
Liver, . .	Not enlarged. Fatty degeneration.	Enlarged. Yellow pigment.

Pathology and Morbid Anatomy.—There is an enormous and sudden destruction of blood-corpuscles in the circulation. This free pigment is mainly eliminated by the kidneys. A portion is stored in the liver, skin, and other situations.

De Haan in the histological examination of kidneys in Java finds that the free hæmoglobin causes injury to the kidneys in the direction of a simple degeneration of the epithelium, or even an extensive nephritis; also, that the excretion of injurious products is interfered with.

The *blood* shows profound alterations. The corpuscles are diminished, there is poikilocytosis, and the corpuscles are very pale.

The *spleen* is enlarged, dark, soft, and diffluent.

The *liver* is enlarged, and yellow on section.

Treatment.—Quinine is useless, and might possibly be injurious.

The bowels should be freely opened as soon as possible.

To allay the vomiting, mustard plasters should be applied to the epigastrium, and the patient should be given ice to suck, and small doses of iced brandy or champagne be administered.

In severe cases nutrient enemata should be given.

If the temperature become subnormal, hot bottles should be applied; hot normal saline intravenous injections (103° F.) may be of use.

For suppression of urine diaphoretics should be employed.

Hearsey recommends—

℞.—Sod. bicarb.,	. . . . .	gr. x.
Liq. hyd. perchlor.,	. . . .	♏xxx.
Aq. chlorof. ad	. . . . .	℥i.

M. F Mist.

Sig.—℥i. every two hours during the first day, and every three hours subsequently till the urine clears.

While for the vomiting he suggests the subcutaneous injection of half a grain of morphine.

CHAPTER XVI.

CHOLERA.

Definition.—An acute, specific infectious disease; of which the endemic home is oriental; and which is characterised by purging and vomiting, cramps, anuria, algidity, and collapse.

It is a disease of high mortality, and is due to the action of a specific intestinal vibrio.

Synonyms — *Cholera Asiatica; Cholera epidemica; Haiza* (Hindustani); *Enerum vandee* (Tamil); *Duba* (Arabic); &c.

History and Geographical Distribution.—Passages in Greek, Sanscrit, Chinese, and Arabic literature are supposed to refer to this disease, but the descriptions are vague. There is no doubt, however, that the disease has been endemic from remote ages in India, and possibly also in China, and other parts of the Further East.

Portuguese, Dutch, and English physicians must have found the disease prevailing in those regions from the fifteenth century onwards, for when a violent *outbreak occurred in Bengal in 1817* it was looked on as an exacerbation of a well-known scourge.

Its story is a simple one. After each periodical violent outbreak in its endemic habitat—India—it has diffused itself more or less widely along the ordinary trade or pilgrim routes of the world.

Each tale of repeated invasion is very much like the other.

Altogether, during the nineteenth century there have been seven European invasions.

1. 1826 to 1839. The disease spread from India through Persia and Central Asia, and reached Southern Russia in 1830, whence it occupied Central and Northern Europe in 1831, the United Kingdom and America in 1832, France and the Iberian Peninsula in 1833, Italy and North Africa in 1834, and gradually wore itself out by 1839.

2. 1840 to 1851.—Our troops carried the disease from India to China, whence it spread by way of Burmah and Central Asia to Persia in 1845, Arabia 1846, Eastern Russia 1847, Europe, the British Isles, and America 1848.

3. 1848 to 1857.—The epidemic began in India where it raged from 1848 to 1850. It spread through Arabia and Persia by land routes and reached Russia in 1852. From 1852 to 1855 the whole of Europe and part of America were again invaded. Our Crimean Army suffered during this epidemic.

4. 1863 to 1867.—Again the usual land trade routes were followed *via* Persia and Arabia to Europe (1865), and Britain and America (1866).

5. 1867 to 1873.—Again by the same route Europe was reached, and from there America. In this epidemic, however, it spread from the latter to the West Indies *via* New Orleans.

6. 1879 to 1887.—Mecca was reached in 1882 and Egypt in 1883.

The Mediterranean littoral was chiefly affected—and the distribution begins to show the influence of increasing sea traffic on the spread of the disease.

This epidemic is also remarkable for the discovery by Koch in Egypt in 1883 of the specific bacillus.

7. 1891 to 1895.—This epidemic, which spread both by land and sea, was one of great rapidity. The sharp outbreak at Hamburg was caused by this epidemic. In 1893, cases occurred at Hull, Grimbsy, and Yarmouth, but England as a whole escaped.

There are certain regions in which, as far as is known, cholera has never occurred. According to *Macleod* these are:—The Andaman Islands, Australia, New Zealand, some Pacific Islands, the Cape, the West Coast, St. Helena, Ascension, the Azores, Bermuda, Chili and Peru, the Orkneys and Shetlands, Iceland, and the Faroe Islands.

Etiology.—The early views as to the specific cause of the disease were many. Some thought atmospheric or telluric conditions responsible; others thought it to be a chemical ferment developing in the soil.

In *1883*, however, the *specific organism was discovered by Koch in Egypt.* He separated the characteristic curved organism from the dejecta and intestines of cholera patients. He showed it to be absent from the blood and viscera, and present only in the intestines where it invaded the mucosa and glands. Proceeding to India in 1884 he examined 72 cases in Calcutta, and found the same organism in all cases. Other observers, as *Finkler*, *Prior*, and others, found somewhat similar organisms in other conditions, but they were found to differ in essential particulars. The lower animals are not susceptible, so that inoculation work is of little value. In some cases intestinal injury may result, and a fatal diarrhœa be set up, but true choleraic lesions do not occur, and the symptoms do not differ from those set up by many other bacteria.

Pettenkofer and *Cunningham* were two great opponents of Koch's theory.

Until about 1892, owing to scarcity of material at home, Koch's views could not meet with general criticism; but on the return of cholera to Europe many observations became possible.

Klein's studies seem to show that other vibrios may be associated with Koch's comma bacillus.

It is now certain, however, that cholera is produced by the vibrionic organism—the comma bacillus.

Among the chief workers on the subject are:—*Gruber*, *Pfeiffer*, *Sanarelli*, and *Metchnikoff*.

The following are details of the comma bacillus, taken from Muir and Ritchie:—

1. They are curved organisms like a German "comma," measuring 1·5 to 2 μ in length, and rather less than 0·5 μ in thickness. They are motile. They have a single terminal flagellum. They do *not* form spores. They readily stain with basic dyes. They are decolourised by Gram.

2. They grow on ordinary media, except potato. The gelatin plate growth is the most characteristic:—

In twenty-four to forty-eight hours minute white points appear which, under a low power, are seen to be irregularly granular. As they become larger their surface looks like fragments of broken glass. Later, liquefaction occurs.

In alkaline broth the organisms grow readily. At 37° C. there is a general turbidity in twelve hours, with a thin surface pellicle.

3. *Cholera-red Reaction.*—If to a twenty-four hours' culture in peptone broth at 37° C. a few drops of pure sulphuric acid are added, a reddish-pink colour is produced. This is due to the fact that *indol* and a *nitrite* are formed by the spirillum. On addition of H_2SO_4 a red coloured nitroso-indol body is produced.

Note.—The *Bacillus coli communis* will form indol, but not nitrite, so the latter must be added before the above result could be obtained with H_2SO_4.

4. *Starch-acid Reaction.*—It was found by *Gordon* that the following diagnostic reaction could be obtained. Cultivate for twenty-four hours at 37° C. in the following medium:—Lemco, 1 gram; peptone, 1 gram; sod. bicarb., 0·1 gram; starch, 0·5 gram; aq. dest. ad 100 c.c., and tinted with litmus.

The comma bacillus will decompose the starch with a strongly acid reaction in the twenty-four hours.

Note.—The Finkler-Prior bacillus only gives a feeble acid reaction by the third day. Streptococci, *Bacillus diphtheriæ*, *B. coli*, *B. enteritidis*, *B. typhosus*, and *B. proteus* all fail to produce any acid reaction in this medium.

5. The comma bacillus is not resistant, being killed in an hour at a temperature of 55° C., and much more rapidly at higher temperatures. They are more resistant to cold, and have been found alive after several hours' exposure to − 10° C. They are quickly killed by most disinfectants. As little as 1 per cent. of lime in water will kill them in an hour.

6. The conditions favourable for growth are warmth, moisture, oxygen, and organic material.

7. Thorough drying, as a rule, kills in two or three minutes. The disease is therefore probably but little dust-borne.

8. The infection will take place by soiled fingers, eating utensils,

food, or drink, any of which may become infected from discharges or other focus, either directly or by the intervention of flies, &c. The comma bacillus has been found alive in the bodies of flies twenty-four hours after feeding on infected material. Their agency is, however, probably always mechanical.

9. None of the lower animals suffer from cholera under natural conditions. Cultures introduced by the mouth gave negative results.

Koch, therefore, neutralised the gastric acidity before the administration, but only one animal out of nineteen showed choleraic symptoms, and this animal had relaxed abdominal walls after a previous abortion. This gave him the idea of interfering with the peristalsis by injections of opium. The subsequent result of feeding with cultures was remarkable, for 30 out of 35 animals died.

With the Finkler-Prior, Deneke, and Miller bacilli a certain, though much smaller, proportion of animals died under like conditions.

10. The intense prostration and collapse are due to toxins elaborated by the vibrios. Toxic phenomena can be produced by the injection of *dead vibrios* into animals. The dead cultures administered by the mouth produce no effect unless the intestinal epithelium is injured, when poisoning may result.

Pfeiffer found that even after heating to 100° C. some toxine remained, and had the same physiological action.

11. *Lazarus* first showed that the serum of patients who had suffered from cholera possessed the power of protecting guinea-pigs when injected in minute quantity with a fatal dose of the organism.

This action of the serum may be present eight or ten days after the attack of the disease, but is most marked four weeks after. It then gradually becomes weaker, and disappears in two or three months.

12. *Pfeiffer's Test.*—A loopful of recent agar culture of the organism to be tested is added to 1 c.c. of ordinary broth containing 0·001 c.c. of anticholera serum. The mixture is then injected into the peritoneal cavity of a young guinea-pig, and the peritoneal fluid of this animal is examined microscopically after a few minutes.

If they have become motionless and swollen (positive result) then the spirilla injected were cholera. If, on the other hand, they are found active and motile (negative result) then the possibility of their being true cholera may be excluded.

13. Other spirilla have been obtained from contaminated water, and sometimes cholera-like symptoms may clinically occur, and other organisms be found. In many of these cases the organism is probably merely a modification of the comma bacillus.

Sometimes rather similar clinical symptoms may be produced by other microbes. The author has seen a case much resembling cholera, even to the rice-water stools, but an examination of which proved it to be an almost pure intestinal culture of *B. coli*.

Summary of Reasons for accepting Koch's Bacillus.—(*a*) It is constantly present in true cases of cholera.

	Koch's Vibrio (*Comma bacillus*).	Metchnikoff's Vibrio.	Finkler and Prior's Vibrio.	Deneke's Vibrio.
'here found,	Human cholera.	Fowl cholera.	Cholera nostras and tooth cavities.	Old cheese.
orphology,	1·5 to 2 μ long. 0·5 μ thick. Comma-shaped. Motile. Single terminal flagellum.	Identical.	Rather thicker in centre and more pointed at ends.	Rather thinner and smaller.
·latine cultures, .	White points, 24 hours. Liquefaction, 50 hours.	Liquefaction, 24 hours.	Liquefaction, 24 hours.	Liquefaction rapid.
olera-red ·eaction, .	Obtained in 24 hours.	Obtained in 24 hours.	No reaction in 24 hours.	No reaction.
irch-acid ·eaction, .	Acid, 24 hrs.		Acid, third day.	
'ects on mimals, .	Subcutaneous inoculation of pure cultures in animals gives no result If exalted, necrosis at point of inoculation.	Subcutaneous inoculation of pure cultures is followed by septicæmia and death.	Result as for Comma bacillus, but modified.	No pathogenic properties.
ato cul-res, . .	No growth at ordinary temperature.	Grows feebly at ordinary temperature.	Grows well at ordinary temperature.	Yellow layer if incubated above 30° C.
iffer's test,	Positive.	Negative.		
narks, .		The fowls have diarrhœa and stupor, and die in 48 hrs. Intestine has a greyish-yellow fluid, sometimes blood-stained.		Sometimes called *Spirillum tyrogenum.* Is a comparatively harmless saprophyte.

(*b*) Known facts of growth, &c., are in conformity with the origin and spread of cholera epidemics.

(*c*) Experiments on animals with the bacillus or its toxines give as reasonable results as one would expect in view of the fact that they do not suffer naturally from the disease.

(*d*) Human experiments and accidental infection strongly favour this view.

(*e*) The specific protecting power of the serum from convalescents is in its favour.

(*f*) Bacteriological methods, assuming Koch's bacillus to be the cause, have been of the greatest value in diagnosis.

(*g*) The results of Haffkine's preventive inoculation (*see paragraph on Treatment*) supply additional favourable evidence.

Symptoms.—These vary somewhat in different epidemics and different individuals.

Incubation Period.—This is from a few hours to five days, generally twelve to twenty-four hours. The disease may be grafted on, and supervene in the course of an attack of diarrhœa, in which case the symptoms suddenly turn to those of cholera.

With such a short incubation period there is seldom time for prodromata. Out of 1,280 cases which have passed (1902-6) through the author's hands, scarcely any instances of "premonitary diarrhœa" can be recalled.

The attack is noticeable amongst all other diseases for its sudden onset, intense symptoms and unsparing hand. Unlike the comparatively limited outbreaks of smallpox, unlike the comparatively low plague mortality amongst Europeans, cholera is a disease which spares but few. Rich or poor, native or Caucasian, young or old are equally liable to this scourge, without doubt one of the most dreadful of deaths.

The following is a clinical picture of a typical case:—The patient is suddenly taken ill with vomiting, and a watery bilious diarrhœa. Within half an hour or so these symptoms become aggravated. The stools become colourless with suspended white flocculi (having much the appearance of rice-water), and are passed involuntarily in large quantities. The retching and vomiting are painful and frequent. Agonising cramps of the legs and abdomen are almost always present and often produce dreadful contortions.

The face, which has an anxious look for the first quarter of an hour, almost immediately becomes pinched and drawn, the cheek bones stand out and the eyes appear as if sunken far into the head leaving great hollow sockets. The skin of the fingers and toes is livid, and has the shrivelled appearance that one associates with prolonged immersion in water (washerwoman's fingers).

The voice is thin and husky; and later becomes no more than a high pitched whisper.

The stage of collapse (algide stage) is reached. The body is cold and clammy. There is complete suppression of urine. The radial

pulse cannot, or can only just, be felt. There is intense thirst, restlessness, and profound debility.

In such a typical case as this, the perpetual rice-water stools, the violent cramps, the agonised sunken hollow face, the shrivelled fingers, will need no cultural or microscopical diagnosis. Once seen, an ordinary case can never be mistaken.

This algide stage terminates either in death or reaction.

When a fatal result supervenes the symptoms continue without abatement, the surface temperature may fall even below 90° F.; and death takes place from apnœa, asthenia, or coma, usually within ten to thirty hours from the beginning of the attack.

If, however, reaction occurs, the pulse gradually returns and the body gets warmer. The patient becomes tranquil and the breathing easy. The temperature approaches normal or is slightly raised; the diarrhœa begins to cease and the stools to become bilious.

The food is retained instead of vomited. After some hours small amounts of urine are secreted, of high specific gravity, albuminous and containing indoxyl. It then becomes watery and more copious.

In a certain proportion of cases such happy return to recovery does not take place. The febrile reaction may become aggravated, and a kind of typhoid state supervene, lasting for some days.

Death may occur during reaction, from complications, such as diarrhœa, asthenia, uræmia or pneumonia.

The earlier cases in an epidemic are generally more severe, and those towards the end milder.

It is often impossible to forsee how cholera will spread.

In some infected ships the author has found that ten or twelve deaths and cases had occurred a day or two before arrival amongst coolie immigrants. Everything would point to the disease spreading, but no other cases occurred after landing all the contacts in quarantine.

Another case the author noted in which a large number of immigrants arrived from a cholera infected port after a voyage of four days. There were no sick on board, but the vessel was detained to complete the five days, on the fifth day a case occurred. Some of the passengers were landed in quarantine and the vessel proceeded to the next port. Five days after the first case, two more cases occurred and were removed. The vessel was detained; and four days later another case (fulminating) was found, so all the contacts were landed at a quarantine station. Here, despite every precaution, the disease spread, and it was a month before it could be finally suppressed.

Other Types of Cholera.—There are two other types of the disease —(1) Ambulatory, (2) Fulminating.

Ambulatory Cases.—These occur during most epidemics, especially towards the end. As the condition is merely one of malaise, and bilious diarrhœa unaccompanied either by cramps or suppression of urine, it can be well understood that such unnoticed cases may form a focus for the further spread of the disease; they have therefore to be very carefully guarded against.

Fulminating Cases.—This term is applied to those cases in which

an acute toxæmia kills the patient before the usual symptoms of diarrhœa and vomiting have declared themselves. The patient usually lies down and dies in a few minutes or else drops down dead. Such sudden deaths during a cholera epidemic will generally be found to be due to cholera and betoken a virulent strain of bacillus. They are, however, rare. Amongst the author's 1280 cases there were only three of this type. Post-mortem, the bowel was full of typical rice-water material.

Sequelæ.—Pregnant women nearly always abort or miscarry.

Anæmia, abscesses, debility, renal or pulmonary complications may occur during convalescence.

Morbid Anatomy and Pathology.—The disease is an acute toxæmia and not a septicæmia.

The comma bacillus is found in no organs or tissues except the intestinal tract.

Rigor mortis is early and prolonged. Post-mortem muscular contractions are not uncommon.

Heart.—The left ventricle is generally contracted and almost empty. The other cavities are dilated with dark fluid blood, with small black threads of clot.

The lungs are small, collapsed, and pale grey in most cases, and the pleural cavities dry and sticky. The *liver*, *spleen*, and *kidneys* are congested.

The *bladder* (in cases dying during the algide stage) was nearly empty, but never quite, in a sequence of 63 cholera post-mortems at the Singapore Quarantine Station during 1903. In cases past this stage a variable amount of urine was found. The supra-renal capsules are dry and sticky.

Bowel.—The peritoneal coat of the intestine is usually dry, sticky, and cream-coloured with injected vessels. The mucous membrane is either dull-red or acutely congested.

The contents of the bowel will be found to vary. If seen very early in the disease the contents will be thick, syrupy, and yellowish-white like Camembert cheese, sometimes suspended in clear watery fluid, the majority being in the small intestine.

At a rather later stage the usual rice-water stools will be seen; the watery fluid either clear or resembling dish-water; and the material equally distributed through the small and large intestine.

In cases where very acute congestion and hæmorrhage have occurred, the appearance presented will be that of a chocolate brown curd suspended in a dirty watery liquid.

In cases dying during the reaction stage there is fæcal pea-soupy matter.

In a few cases the bowel will be empty, but for viscid albuminous material between the rugæ.

The *case-mortality* varies from 40 to 90 per cent. It is very seldom below 60 per cent.

Treatment.—There is no specific drug for use in cholera.

During epidemics astringent and sedative drugs are frequently

enjoined for diarrhœa. Most of these, such as chlorodyne, lead and opium, chalk and opium, opium pill, sulphuric acid and laudanum, are highly dangerous. Koch has proved that even if a certain natural immunity exists it is immediately abolished directly the peristalsis is interfered with by opium.

For the actual treatment many drugs have been recommended, but are now merely of historical interest:—Chloroform, atropine, nitroglycerine, calomel, carbolic acid, bismuth, *Strophanthus*, *Cannabis Indica*, turpentine, eucalyptus oil, &c., are among the lengthy list.

So far only one drug has been found which has a definite effect during the algide stage and that is—salol.

If drugs are to be given at all, give the following:—

℞.—Salol,	gr. x.
Mucilage,	ʒi.
Sp. chlorof,	♏xx.
Water,	ʒi.

M. F. Mist.

Sig.—ʒi. every two hours until the reaction sets in. Then

℞.—Sod. bicarb.,	gr. x.
Sp. chlorof.,	♏xx.
Sp. æth. nit.,	♏xx.
Aq. ad.	ʒii.

Sig.—ʒij. every four hours till urinary secretion is thoroughly established and normal.

Stimulants will be needed throughout; and, if they cannot be retained by the stomach, should take the form of hypodermic injections of brandy or ether.

Warmth should be secured by hot bottles.

Nourishment.—Nothing but iced whey, iced champagne, or iced brandy and water should be given during the algide stage.

THE MOST RATIONAL OF ALL METHODS OF TREATMENT is that adopted notably by *Cox* of Shanghai, who combines such general treatment as given above, with large intravenous injections of warm saline fluid. When it is considered that the toxæmia has resulted in the loss (up to the extreme limit) of body fluid, the advantages of such treatment will be obvious.

With the vital functions at such low ebb it is useless to give either rectal or subcutaneous saline injections.

The mixture should be made by dissolving 100 grs. of sodium chloride in each quart of distilled water. Raise the temperature of this to 100° F., and inject some 2 or 3 quarts, slowly, into a vein by the gravity of a 2 or 3 feet head of water. This should be continued till the pulse is restored. It may be necessary to repeat the procedure if collapse is again imminent. By this means many moribund cases have been saved.

During convalescence, the return to solid food should be careful and gradual.

Prophylaxis.—Great care should be exercised in preserving the general health. Fatigue, chills, and excesses should be avoided; unripe and over-ripe fruit should be steadfastly shunned during epidemics; nor should water be taken unless specially boiled and filtered.

Eucalyptus oil has a definite prophylactic effect.

In an epidemic on the Singapore Quarantine Station in 1903 the following results were obtained:—

Eucalyptus oil ♏x. twice daily was administered to—

(*a*) Five out of seven workmen in a hut near cholera contacts. The two who did not take it were alone attacked.

(*b*) Three batches of 50 in three badly infected wards, for one week. During that period only one case occurred amongst these men, as against six cases amongst the remainder.

In another epidemic in the same place in 1906 the author put some 400 to 500 contacts on eucalyptus oil with the result of immediately lowering the incidence in a very marked degree.

Haffkine's Anti-cholera Inoculation. — The virulence of the organism is attenuated by passing sterile air over the surface of the cultures.

The virulence is exalted by continued passage through the peritoneum of guinea-pigs.

Subcutaneous injection of this exalted virus produces a local necrosis, often fatal.

If, however, the attenuated virus is first injected, the subsequent injection of exalted virus only produces a local œdema.

By this progressive injection, a high degree of immunity is secured against every method of inoculation of the organism, either into the tissues or by the mouth.

Haffkine began his method on the human subject in India in 1894.

Two and sometimes three inoculations are made with the attenuated virus before the *virus exalté* is used.

Up to 1895, 70,000 injections had been made in 43,179 persons, with fairly encouraging results.

Haffkine subsequently found that, in man, local necrosis was not produced by primary injection of exalted virus. He therefore abandoned the preliminary use of the attenuated virus.

Powell's results in Assam are:—

	Coolies.	Cases.	Death.
Inoculated, . .	5,778	27	14
Non-inoculated, .	6,549	198	124

CHAPTER XVII.

DENGUE.

Definition.—A specific febrile disease peculiar to warm climates, and characterised by severe articular and muscular pains, and a well-marked eruption.

Synonyms.—*Exanthesis rosalia: dandy fever: break-bone fever: colorado: fièvre éruptive rhumatismale*, &c.

The name of the disease—dengue—is derived, according to Hirsch, from the Spanish equivalent of "dandy"—from the stiff and dandified carriage of the sufferer.

Geographical Distribution.—There is no mention of the disease before 1780.

Pandemics have occurred on at least three occasions. From 1780 to 1783, it spread from India to Java, to Egypt, to Zanzibar, to Europe, and probably to America. From 1824 to 1828 it was widespread in India, Burmah, North and South America, and the West Indies. The last pandemic lasted from 1870 to 1873, having begun at Zanzibar, and spread to Port Said, India, Siam, Burmah, Java, Sumatra, Mauritius, and then to the United States.

Since this time the distribution has been less widespread, and has only appeared in limited epidemics.

It is to be found in almost all tropical countries—perhaps most frequently of all in the West Indies (*Manson*).

Etiology.—Although from its behaviour and spread, dengue must certainly be due to a microbe, yet the specific germ has not yet been found. Several observers have discovered what they thought to be the parasitic cause, but without satisfactory demonstration. In 1902 Graham described an amœba resembling Pyrosoma bigeminum in the blood of his dengue patients at Beyrout in Syria, and his experiments tended to show that a species of *Culex* might act as an intermediate host.*

It is essentially a disease of warm climates—and seems to frequent coast districts, showing, as a rule, but little tendency to spread inland. A possible solution might be found in the distribution of a certain species of mosquito or other intermediate propagating host.

In one epidemic seen by the author the only mosquitoes present were *Stegomyia fasciata*. These are day feeders and very fond of maritime districts.

The *incubation period* has been variously placed at one to seven days. The rapidity with which the disease spreads would seem to point to the shorter of these periods as being more likely to be correct.

* Quoted by Manson from *Med. Rec.*, N.Y.

Race and Sex Distribution.—No race enjoys immunity. In some outbreaks natives, such as negroes, Indians, and Chinese, have suffered more than the neighbouring European residents. The youngest ages are not exempt, and both sexes seem equally susceptible.

Susceptibility tends to be exhausted by one attack, but second and third attacks are not at all uncommon.

Symptoms.—The prominent symptoms are fever, pain, and cutaneous eruptions. The clinical features are distinctive.

There may be a little malaise, or a few prodromal pains, or occasionally a rigor, but the onset is usually very sudden. Without warning, the patient is seized with severe *muscular and arthritic*

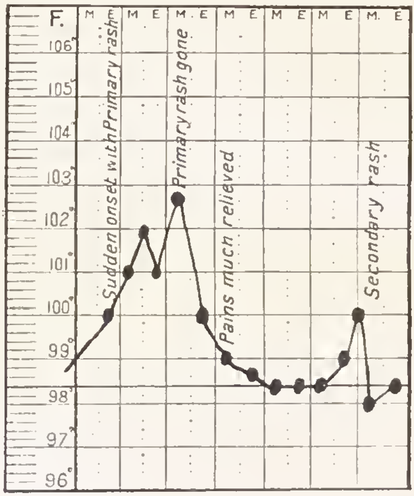

Fig. 35.—Chart illustrating case of dengue.

pains. The occurrence of *fever* is practically synchronous with the pain, and either immediately, or within an hour, the *primary rash* makes its appearance. This rash is an erythematous one, somewhat resembling scarlet fever, and is chiefly confined to the face, and the upper and lower extremities. It is frequently very transient, and at most does not last more than twenty-four hours. Meanwhile, the urgent symptoms increase, the headache is severe, the pains become very acute, and the temperature continues to rise, occasionally to 105° F., or thereabouts. The tongue is coated with a white fur, the sense of taste is vitiated, and anorexia is usually the rule.

About the third day of the fever, a crisis usually occurs, and with the decline of fever great relief is experienced; the pains and headache are relieved, and, in the course of one or two days, the temperature reaches the normal.

At about the fifth to seventh day from the commencement of the attack there is a slight return of fever. This is usually never very high, and may occur at night, thus escaping notice.

With this secondary rise of temperature a secondary eruption takes place, this time of a rubeoloid type, often somewhat resembling measles. It is chiefly confined to the extremities, and is either of a few hours duration, or else may persist for a day or two. A recurrence of pains for a few days is generally met with.

Convalescence is soon established—the tongue clears, the appetite returns, and the patient generally feels himself again in a few days.

There is a slight desquamation of a furfuraceous nature when the

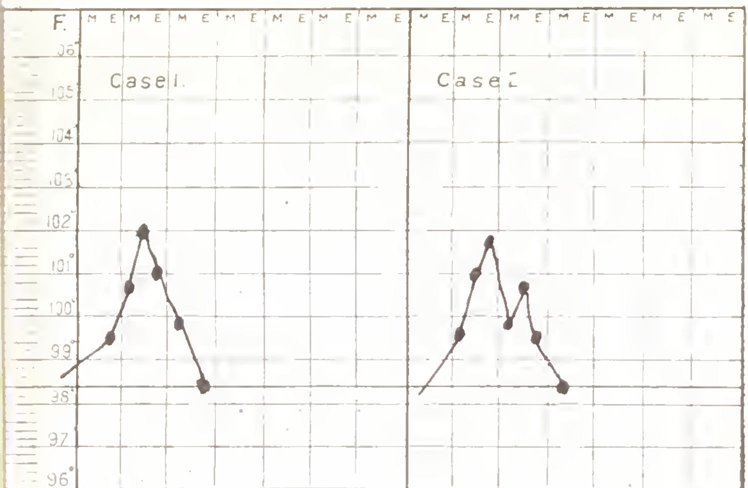

Fig. 36.—Charts of two cases of pseudo-dengue occurring in the same house at an interval of one week.

secondary rash has been at all marked. If the rash has been but slight, the desquamation is scarcely noticeable.

Such is the usual course of a typical case. In every epidemic will be seen graver as well as slighter cases.

The accompanying chart from the author's practice is fairly typical of an ordinary attack. (Fig. 35.)

Sequelæ, &c.—In very many cases pains of a rheumatic nature persist for some months, and are very hard to cure. Attacks of mental depression, sleeplessness, crops of boils, lymphadenitis, and orchitis are occasional sequelæ.

Nephritis has not been observed. Miscarriage is rare. Although relapses are not uncommon, yet, on the whole, the disease may be

classed as a mild one, although extremely painful and disagreeable to the patient himself. The *mortality* is almost nil.

Post-mortem records are consequently few. Some pulmonary and intercranial inflammations were the features of four post-mortems made by Nogue in Cochin China in 1895 (*Manson*).

Diagnosis.—The presence of articular and muscular pains should serve to differentiate dengue from the other exanthemata.

The presence of a rash will preclude rheumatic fever or influenza. Dengue has many analogies to influenza—in fact, it has been called the influenza of the tropics—and many cases of feverish cold and influenza occurring in the tropics are hastily diagnosed as dengue. This is, of course, incorrect. *The rash is as much a part of dengue as it is of smallpox or measles*, and those cases in which no rash occurs should have the diagnosis of dengue reconsidered.

The two accompanying charts (Fig. 36) illustrate some of these pseudo-dengues. Two cases of feverish cold occurring in the same house; the incubation probably one week, and the symptoms those of a cold with some aches and pains—a foul tongue, constipation, and slight headache. Bacteriological investigation in such cases would probably give either Friedlaender's bacillus, or possibly *Bacillus coryzæ segmentosus* or *M. catarrhalis.*

Treatment.—Light diet, rest in bed, and sufficient warmth should be secured during the attack. A purge should commence the treatment, which may be followed by some antipyretic such as phenacetin or antipyrin.

The author has found the following to be of the greatest value, not only for lessening the acute symptoms, but also for mitigating the subsequent rheumatic pains :—

℞.—Quin. salicyl., ... gr. v.

Sig.—One such powder to be taken in cachet every four hours.

Or, ℞.—Aspirin, ... gr. vii.

t. d. s.

Or Carne Ross' influenza treatment may prove useful, which consists of Ol. cinnamon, ten drops every two hours till temperature is normal, when the amount should be diminished to ten drops three times a day.

In all these cases, however, although we may lessen the urgency of the symptoms, and perhaps slightly curtail them, yet we cannot obviate the definite course of the disease.

Ordinary measures should be taken for any special symptoms which arise. Hyperpyrexia will demand cold sponging. Excessive pain may require a hypodermic of morphia.

Alcohol is not generally indicated.

During convalescence a tonic, such as compound syrup of the hypophosphites, is indicated, with occasional massage, and possibly change of air.

CHAPTER XVIII.

DIATHERMASIA AND PHŒBISM.

DIATHERMASIA.

ONE of the most remarkable facts to do with man is the capability, when in health, of being able to maintain his normal body heat under extreme conditions of surrounding temperature.

The body temperature is a matter of reflex nervous influence, and it is the difference in the reaction of their tissues to external thermic influences that constitutes the essential distinction between the warm- and cold-blooded animals. We have no evidence to show when the necessary nervous mechanism was evolved by the former, but we know that the temperature of the so-called cold-blooded animal is always a fraction of a degree above that of their environment, and that at some especially active vital periods (*i.e.*, sexual) it may be several degrees higher. This heat is mere waste—a bye-product—and it is easy to conceive how natural selection may have made use of this unavoidable conversion of chemical energy into heat, and turned it to sufficient account to enable the organism to modify its heat production and heat loss in accordance with its requirements, and thus protect its tissues from the tyranny of their environment, until such state is reached when five-sixths of chemical intake energy (in the form of food) leaves the body as heat, and only one-sixth as mechanical energy.

The thermotaxic centre which regulates the body heat is probably situated in the optic thalamus.

There are three sets of nerves which control the heat production and discharge:—

1. Vasomotor nerves. . . .	Discharge.
2. Secretory nerves of sweat glands,	
3. Trophic nerves.	Production.

The thermotaxic mechanism is about the latest evolved of the automatic nervous mechanism of the human body; thus it is the latest to be unfolded in the child, a fact explaining the well-known irregularity of a child's heat phenomena. European and native children in the tropics are therefore very much on a par during the first few years of their lives, as regards their thermotaxic mechanisms; and it is not until the European child's progressive

development causes its thermotaxic mechanism to approximate to the adult type, that it begins to be at a disadvantage. When this does occur, a portion of the nervous energy which ought to be devoted to evolution and growth is wasted, as in the case of the adult European, but with more disastrous results.

In the discharge of CO_2 we have a gauge of tissue waste consequent on the setting in action of that section of the thermotaxic mechanism which regulates the loss of heat. This thermotaxic mechanism is, in the European, "set" at a different point to that of the native.

In the European, the discharge of CO_2 at—

4° C. (39·2° F.)	is	210	grammes per six hours.	
9° C. (48·2° F.)	,,	192	,,	,,
16° C. (60·8° F.)	,,	158	,,	,,
24° C. (72·2° F.)	,,	166	,,	,,
30° C. (86·0° F.)	,,	170	,,	,,

The point of minimum CO_2 production in the European is therefore at about 60° F. In the tropical races this CO_2 index is certainly higher than in the European, and various considerations indicate that it must be somewhere near the daily mean temperature of the regions where the various races have long been existing.

Having grasped this point two important inferences may be drawn with reference to the action of a tropical environment on the heat mechanism of a European:—

1. It is evident that the *section* of the European's thermotaxic mechanism *which regulates the loss of heat* is called into action much more energetically and continuously than is natural to it—*i.e.*, it is subject to a continual strain.

2. It is no less evident that the *heat-producing section* of the European's mechanism is called into action to a very much less extent than natural; its large resources lie dormant, and may be a potential source of danger to their owner.

A consideration of the foregoing remarks, and of these two propositions, will throw much light on the health of our countrymen in the tropics, and on the various *heat* effects which have been called by so many terms, and will obviate the necessity for seeking the "specific" origin, which has been claimed by some.

There is one fact which stands out clearly, and that is, that the European who is subjected to a continual atmospheric temperature higher than that to which he is normally accustomed, or who is subjected to abnormally high atmospheric temperature, either in or out of the direct sun's rays, must have his thermotaxic mechanism profoundly modified (assimilating to the native type), or else severely strained, and soon broken down.

In the former case the European becomes acclimatised more or less completely; in the latter case he exhibits various pathological phenomena ranging from febricula to diathermasia.

In either case there must be an intermediate stage preceding the

inal one of acclimatisation or of break-down; this intermediate tage being either short or long, and presenting various phenomena. t is a period of strain. The characteristic effect of strain on any iving tissue is to produce a condition of irritable weakness: a condition in which the tissue, on the one hand, reacts with abnormal eadiness to external impulses, and, on the other, does so with less orce than natural.

This condition of the thermotaxic mechanism is evidenced by ever of a variable and indefinite character (*Febricula*); it is a ondition in which the regulative grasp of the central nervous ystem on the metabolism of the tissues is slackened and fitful: at ne time the grasp is tightened, and the heat phenomena return to he normal condition; then it is loosened, and tumult takes the lace of order.

If its grasp be too long, or two suddenly loosened, it may be impossible for the nervous system to recover command, katabolism runs iot, overpowering the other section of the thermotaxic mechanism, he temperature of the body rises rapidly (*Diathermasia*), and death nay occur from hyperpyrexia.

Having thus worked over the ground leading up to it, we can etter consider the subject from the usual routine points of view:—

Definition.—Diathermasia is an acute dyscrasia, due to the ffect of heat on the thermotaxic mechanism of a non acclimatised r predisposed person, and characterised by hyperpyrexia and pulnonary congestion.

Synonyms.—Heat stroke, insolation, heat apoplexy, thermic ever, siriasis.

Geographical Distribution.—The following regions are those 1 which the condition is most often found:—The Red Sea, Persian ulf, Sind and Bengal provinces in India, Lower Burmah, parts of lgeria, the valleys of the Nile, Mississippi, Amazon, and La lata rivers, and the hotter parts of the Eastern littoral of the nited States and of Queensland. It may also naturally occur in ny place having the necessary thermal environment combined with ne requisite personal factors. It is unknown in Europe, nor is it nown above an altitude of 600 feet (*Sambon*).

Etiology.—The condition is brought about by the effect of eat, causing a loss of control by the central nervous system over ne metabolism of the tissues, paralysing the nerve sections of the iermotaxic centre which have control of heat discharge (vasomotor nd sweat glands) by the hyperactivity of the heat-producing section rophic nerves). Non-acclimatisation will naturally predispose to is condition, and, therefore, new-comers and Europeans will be ore liable than old residents or natives.

All ages and both sexes are susceptible, although, in consequence their habits and frequent exposure to predisposing causes, men e more liable to diathermasia than women.

Other predisposing influences will be intemperance, fatigue, and bilitating diseases.

Symptoms.—There are generally some *prodromata* for a few hours before an attack. These usually take the form of lassitude, headache, or giddiness; occasionally there may be thirst, apprehension, or even vomiting; and Longmore mentions a frequent desire to micturate as a common symptom. Not infrequently the attack comes on during the night.

Whether with or without prodromata the condition becomes very quickly serious. The temperature mounts rapidly, reaching occasionally 110° F. There is either coma or delirious insensibility.

The face is flushed or cyanosed; the conjunctivæ injected; the pupils may be contracted or dilated.

The skin is hot and dry, and occasionally may have a mousey odour. The pulse is rapid and of low tension, soon becoming intermittent and thready.

The respiration is usually embarrassed and heaving, occasionally stertorous.

Just before death tetanic convulsions are not uncommon.

The urine is scanty, and contains albumin and casts.

The superficial reflexes are very sluggish.

The cause of death may vary. If the attack is complicated by cerebral hæmorrhage, that of itself may ring down the curtain. On the other hand, death may be due to tetanic contraction of the ventricles (*Lauder Brunton*) or respiratory failure (*Manson*). If death does occur, it is usually a matter of hours from the onset of the attack.

Sequelæ.—If recovery takes place there are practically no sequelæ unless it has been complicated by cerebral hæmorrhage, when the usual resultant symptoms will be present. There is almost always, however, an epicritical increase of urea.

Prognosis.—The case mortality in diathermasia is very high, probably about 75 per cent. It is necessary that prompt and vigorous action be taken if life is to be saved.

Pathology and Morbid Anatomy.—Rigor mortis is early. The skin is often mottled, showing purplish discoloration.

The blood is remarkably fluid, and exhibits usually an acid reaction, as also, it is stated, do the muscles.

Head.—The sinuses of the dura mater will be found engorged with dark fluid blood, and the vessels of the pia mater much injected. The whole brain is highly vascular, bleeding freely wherever incisions are made. The ventricles usually contain serous fluid.

Heart.—This organ usually shows remarkably rigid rigor mortis, while the cavities contain fluid blood, with little or no clot.

Lungs.—On section, the lungs will be found intensely engorged with dark fluid blood, which escapes from the cut surfaces.

Liver is congested.

Spleen.—No enlargement is usually found beyond what would be expected in a highly-engorged organ.

Kidneys.—These organs are markedly congested, the pyramids standing out prominently and being of a very dark colour.

Stomach and Intestines.—These show, as a rule, no pathological appearances.

Diagnosis.—The presence of considerable pyrexia will differen-iate diathermasia from uræmic, diabetic, alcoholic, or narcotic coma.

Cerebral malaria must be excluded, for which a microscopical xamination of the blood may be necessary, aided by any history btainable and by percussion of splenic dulness.

Again, the retraction of the head in cerebro-spinal fever, together vith a not unduly high temperature, irregular pupils, and long uration should preclude the possibility of a mistake in this direction.

Treatment.—It is important that immediate attention should e given to the reduction of the hyperpyrexia.

Quinine is of no use if malaria has been excluded.

The antipyretic drugs are dangerous to the already enfeebled heart.

Strychnine should be avoided, owing to the tendency to tetanic nuscular contractions.

The patient should be at once placed in a cold bath, and ice pplied to the head; or the body may be packed with ice.

A hypodermic of $\frac{1}{75}$ gr. hyoscine hydrobromide should be given t once to quiet the intense cerebral hyperactivity.

If, or when, the pulse becomes irregular and thready, a hypo-ermic of $\frac{1}{100}$ gr. digitalin should be administered, and repeated in quarter of an hour, if necessary.

When the temperature has fallen to 103° F. the cold applications iould be discontinued, and the patient wrapped in a blanket with ot bottles By this time, very likely, the grasp of the central ervous system on the thermotaxic mechanism will be becoming estored, and gradually equilibrium be established between the hannels of heat production and discharge, resulting in the action of ie sweat glands and a reduction of the temperature.

Occasionally, however, instead of diaphoresis occurring, the emperature will rise again rapidly, and may, in spite of renewed old-pack or other treatment, pass beyond control, and end in the eath of the patient.

If, however, profuse diaphoresis does occur, and the temperature ılls to nearly normal, it may be necessary to administer alcohol; ut it should be exhibited with caution, and only if the state of the ulse urgently indicates it.

Great care should be taken during *convalescence* to prevent relapse or recurrence. The patient should be kept as cool and uiet as possible; alcohol should be avoided; the diet should be utritious, but light.

The bowels should be kept open: small doses of bromide may be iven for a week or so.

R.—Ammon. brom., ...	gr. x.
Syr. aurantii, ...	ʒi.
Aq. ad	℥i.

M. F. Mist.

Sig.—ʒi. t. d. s.

Following this, a change to a cooler climate should be ordered, ith a month's course of hypophosphites.

PHŒBISM.

Whereas, in diathermasia, we have to do with the effects of the long wave length and low frequency red and infra-red spectrum rays, so, in phœbism, we have to reckon with the short wave length and high frequency actinic rays, comprising the blue, violet (down to a wave length of 15 millionths of an inch), and ultra-violet rays of the spectrum.

A certain consideration of the generally accepted principles of actinic light is a necessary prelude to a review of its material manifestations and pathological effects.

Theory of Light.—The most probable hypothesis of ordinary light is that of Stokes, who considered it as a series of waves of transverse vibrations, each series consisting on the average of at least 50,000 successive vibrations, in which each ripple is one of a series that gradually dies away. As the series of waves is complete, it can therefore undergo regular refraction and reflexion.

All kinds of light in the visible spectrum are comprised between the extreme red at the one end and the extreme violet at the other. Their wave lengths vary between about 32 millionths of an inch (extreme red) and 15 millionths of an inch (extreme violet).

Ultra-violet Rays.—But besides the waves of various colours, between those limits, which are visible, there are others which are physically identical but yet invisible, and these extend both below the extreme red and beyond the extreme violet. They are not obvious to us in the ordinary course owing to our very limited range of visual perception, but these invisible rays are none the less "light," since they can be reflected, refracted, polarised, and diffracted.

Ultra-violet light may be obtained from an arc lamp, especially if one of the terminals is made of zinc or aluminium, the light from these substances being very rich in ultra-violet rays. There are a considerable number of these rays in sunlight, although it cannot be called rich in them, as so much is absorbed by the upper regions of the atmosphere.

In addition to these ultra-violet rays of known properties, we have several other rays, also invisible, and somewhat analogous.

Röntgen Rays.—On 8th November, 1895, Professor Röntgen, by means of some barium platino-cyanide paper, found that cathode rays in a high vacuum tube emitted a certain kind of ray which penetrated materials hitherto considered impermeable to light. A sheet of aluminium $\frac{1}{20}$ inch thick, though opaque to every other previously known kind of light, is for this kind of light practically transparent. The nature of these Röntgen rays is not incompatible with Stokes' theory, if they are considered to consist of solitary ripples (and not of a series of waves as in ordinary light), each of not more than one or one and a-half waves.

According to him, the Röntgen light is generated at the anti-cathode by impact of the flying negatively-electrified molecules

which constitute the cathode stream. At the moment when the flying molecules strike and rebound there will be a quiver in their electric charge; in other words, the charge on the molecule will perform an oscillation.

This oscillation, being of extremely short period, and dying out after about one or two complete periods, will generate a wave, which, though of a frequency as high as, or even higher than, that of ordinary ultra-violet light, and therefore capable of producing kindred effects, will not be capable of being made to interfere, nor to undergo regular refraction or reflexion, because it does not consist of a complete train of waves.

In sunlight there do not appear to be any Röntgen rays, nor yet in the light of the electric arc; for neither of these sources contains any rays that will affect a photographic plate that is protected by an aluminium sheet.

Other somewhat similar rays should be mentioned.

Becquerel's Rays.—In 1896 Becquerel and Thomson independently discovered the radiations emitted by the salts of the metal uranium. Many of these, whether in the dark or in the light, emit a sort of invisible light, which can pass through aluminium, and produce on a photographic plate shadows of interposed metal objects. This effect appears to be due to an invisible phosphorescence of a persistent sort. Just as luminous paint goes on emitting visible light for many hours after it has been shone upon, so these substances go on month after month emitting an invisible light. Hence the phenomenon is known as one of hyperphosphorescence. Becquerel's rays possess, like ultra-violet light, and like Röntgen's rays (though to a less degree), the property of diselectrifying charged bodies. These rays are absorbed by air. Water is transparent to them.

There appears to be no doubt that the uranium rays are a species of extreme ultra-violet light, having a wave length certainly less than 10 micro-centimetres, and a frequency certainly greater than 3 000 billions per second.

Phosphorus Light.—The pale light emitted by phosphorus when oxidising in moist air is accompanied by some invisible rays which will penetrate through black paper or celluloid, but will not pass through aluminium.

Glow-worms and Fireflies.—Muraoka, in Japan, using 1 000 fireflies in a shallow box, over a screened photographic plate, found that they emitted rays which, after filtration through card or copper plates, will act photographically (Thompson).

Other rays, such as Wiedemann's rays, Thompson's paracathodic and diacathodic rays, and Goldstein's rays, do not call for mention in such a chapter as this.

Thus far we have seen that there are certain actinic rays at the violet end of the spectrum, with invisible rays continued in the ultra-violet.

Although these ultra-violet rays are of too small a wave length to

affect our sense of sight, and although we know but little of the most extreme rays, yet those which we know possess several remarkable properties.

Properties of Ultra-violet Rays.—I. They produce photochemical, *chemical*, and photographic *effects*, some of which are also produced by visible light, especially by the blue and violet waves. For instance, if hydrogen and chlorine are mixed in a bulb of violet glass (which allows only the violet and ultra-violet rays to pass), and the light from an arc lamp is concentrated on to it by a quartz lens (which is transparent to ultra-violet rays), an explosion of chemical combination takes place, showing that the ultra-violet rays of sunlight are the active agent in causing the old-time experiment.

Again, if a quartz prism and lens are used to get a spectrum, and if a long strip of photographic sensitised paper is stretched along the length of the visible spectrum, and some distance beyond, and exposed for several minutes, it will be found to have darkened at the violet end and also for some distance beyond, where our eyes see nothing.

These actinic rays have, therefore, pronounced chemical action.

II. They produce, also, certain *physiological effects* on animal and vegetable tissues.

Macleod, writing on melanosis from exposure to light rays, says:—"The exposure of the skin to strong sunlight causes melanosis, which may take the form of freckles or a more diffuse sunburn, and it is the primary cause of the dark complexions of the inhabitants of sunny climates. This pigment has been described as a provision of nature against the penetration and harmful rays of the spectrum—namely, the violet, ultra-violet, and blue rays—and not to the red heat rays. In it the melanin granules are increased in and between the cells of the epidermis, and pigmentation occurs in regions where it is not generally present."

Crocker, in speaking of the "Finsen" treatment, says:—"Sunlight, where it is available, or, in its absence, the electric arc light, is concentrated by means of an apparatus which cuts off the heat rays, and leaves only the actinic—viz., the blue, violet, and ultra-violet rays—to act upon the diseased tissue, from which the blood must be pressed out, as the red corpuscles in the skin prevent the deep action of the rays. . . . No effect is seen at first, but after from twelve to forty-eight hours inflammatory action sets in, with redness, swelling, and sometimes bullæ, or oozing of the part directly exposed to the rays."

Stelwagon, discussing X-ray dermatitis, describes a usual latent period of several days after exposure, followed by an erythema lasting for several days or weeks. In other cases it is accompanied by vesiculation and puffiness, with symptoms of irritation and a persistent course. Less frequently sluggish ulcers, with an inflammatory border, may develop, defying treatment. He goes on to say:—"The pathology of the malady is not clearly understood; many observers believe that the cutaneous disturbances are not

rimarily ascribable to local action on the cells of the derma, but hat they are rather of a tropho-neurotic nature, due to neuritis; nd this, according to Oudin, Barthélemy, and Darier, is not a eripheral neuritis connected with the dermic nerve terminals, but probably at first central, during the period which might be called hat of the incubation of the phenomena, to become subsequently entrifugal, and to manifest itself by distinct alterations of nutrition."

III. In addition to the chemical and physiological effects, we ave *phosphorescent effects*. Many substances have the property of bsorbing the very short rays of ultra-violet light and transforming hem into rays of longer length that are visible to our eyes.

For instance, if a lantern be taken with a quartz lens, and covered ith a cap of dark violet glass to cut off all the visible light except little violet that unavoidably accompanies the invisible ultra-violet ys, and this beam of invisible rays be directed upon a cube of ranium glass, the waves become visible as a brilliant green. bottle of paraffin oil becomes brilliant blue, &c.

IV. Lastly, the actinic rays have certain *electrical properties*. rofessor Hertz discovered that ultra-violet light will produce selectrification of electrified bodies, but with a limitation. If the ectrified surface is that of a metal surrounded by air, then when tra-violet light falls upon it, it will produce diselectrification if the rface is *negatively* electrified, but not if positively electrified.

Rontgen rays, on the other hand, will produce a diselectrification hether the charge be positive or negative.

In reviewing the whole subject, therefore, certain acts stand out amid much that is hazy.

(*a*) There are ultra-violet rays in sunlight, but no Röntgen rays.

(*b*) The violet and ultra-violet rays produce chemical, pathological, osphorescent, and electrical phenomena.

(*c*) The red blood-corpuscles in the skin tissues are one of nature's eens to ward off the harmful penetration of actinic rays.

(*d*) That excessive exposure calls out on the part of nature, an ditional safeguard—secondary melanosis.

(*e*) There is a primary melanosis in the case of indigenous habitants of hot climates.

(*f*) That the continued application of ultra-violet rays seemed to ve not only a local action on human tissues, but a central action, h secondary trophic lesions.

We are now in a position to consider by routine methods the bject of phœbism.

Definition.—Phœbism is a condition akin to shock, caused by inic solar rays, characterised by meningeal congestion, and owed by cerebral or cerebro-spinal disturbances.

Synonyms.—Sunstroke, insolation, coup de soleil, sun amatism.

Distribution.—A condition due to the action of actinic solar s; it may occur anywhere; but, from the nature of things, will usually a matter of the most tropical regions.

Etiology.—The continued local application of ultra-violet rays has not only a local inflammatory action, but a central action, with secondary trophic lesions.

From the symptoms of phœbism, as well as from the post-mortem conditions found in fatal cases, we conclude that the condition is one of cerebral shock caused by exposure to these portions of the solar rays, and perhaps also to the actinic portion of the visible spectrum.

The *modus operandi* of this cause is probably primarily an acute cerebral or cerebro-spinal congestion, followed by a chronic inflammatory condition of the meninges.

Conditions, in many severe cases, point also to some obscure chemical or electrical action on the nerve centres by these actinic rays, such as would account for the sudden deaths during exposure to the sun.

That these rays are capable of both chemical and electrical action, we have already seen.

Newcomers and Europeans are more susceptible than natives.

As in the case of diathermasia, it would seem that a habit of acclimatisation can be acquired by gradual habituation. Such immunity is probably acquired hereditarily by natives.

All ages and both sexes are susceptible, but, from the nature of their habits, men are more subject to phœbism than women.

Symptoms.—The symptoms of phœbism will naturally vary according to the concentration, area, and duration of the actinic exposure, as also to the degree of acclimatisation enjoyed by the person attacked.

The milder cases are familar to all.

One class of case is that of a person working, with his coat off, in the tropical sun, his head being duly protected by a topée. Within an hour or so of the first exposure, he gets what is called sunburnt—*i.e.*, a diffuse erythema occurs—a pattern may be outlined on the skin where the seams or hems of the shirt had formed a double layer, and offered more protection; and some subcutaneous exudation may also occur, amounting sometimes to a painful œdema. The skin subsequently peels, and a certain amount of pigmentation may remain. In these cases there is a slight shock, depending on the extent of the superficial area thus exposed. A slightly febrile reaction occurs later, and may amount to several degrees F. if the erythema is widespread. A headache and feeling of lassitude often accompany the reaction stage. The condition, however, readily answers to a purge and a day or two's rest, although the affected skin may be too sore to touch for several days, and may demand local medication.

Another class of mild case is exemplified by the casualty list on a hot summer day amongst an open air sight-seeing crowd, at home; also at tropical race-meetings, military parades, &c.

In these cases the clothing is generally sufficient to obviate a superficial dermal effect by the ultra-violet and other actinic rays.

The condition appears to be rather one of slight, but concentrated, action on the less well protected skull, temples, or nape of the neck, which are so much exposed by improper headgear, or by the reflection of the rays upon the temples from the bright surface of the ground, or of the sea, &c.

The symptoms are a sudden feeling of giddiness and faintness, which may amount to syncope. The use of a cool darkened room, with the application of cold to the head, and smelling salts to the nose, will usually prove sufficient treatment. A slight headache may persist for a day or two, with or without temporary disorders of hearing or vision.

The effects of these rays upon persons much exposed to a hot tropical sun, may be a much more serious matter, amounting to what has been called by writers, insolation (*Fayrer*), or sun traumatism (*Manson*).

The concentrated actinic action may produce profound shock to the nerve centres, causing almost instantaneous death from heart or respiratory paralysis.

The following is, however, the usual train of symptoms in severe cases of phœbism:—

A person is much exposed to the sun. He suddenly loses consciousness. The pulse is small and of low tension; the skin is moist. The temperature is normal or sub-normal. The breathing is laboured—occasionally stertorous. The condition is one of shock and cerebral hyperæmia, with accompanying congestion of the meninges.

At this stage the condition may (1) either tend towards convalescence with complete recovery, and no injurious after-effects; or (2) develop into acute meningitis; or (3) develop into a chronic inflammatory condition leading to the thickening of the calvaria, obliteration of the diplöe, flattening of the convolutions, and accompanied by cerebral disturbances, such as tremor, amnesia, amaurosis, deafness, paresis, epilepsy, and dementia.

Treatment.—The treatment of phœbism resolves itself into the removal of the patient to a cool dark room; shaving the head, and applying cold to the scalp.

A preliminary aperient is advisable, and the food should be light and digestible.

Other symptoms should be treated as the occasion demands.

Cardiac failure may require hypodermics of digitalis, and precordial applications of heat.

Restlessness and insomnia may indicate the administration of bromides.

An early removal should be advised to a cold climate, directly the patient is fit to travel.

In the cases of types 2 and 3 (above) the patient should be told to avoid any subsequent return to the tropics after leaving.

The following brief notes of cases mentioned by *Fayrer* may be of interest, showing the usual course of cases of phœbism:—

CASE 1.—Survey offieer, æt. 27. In Beluehistan in 1889, was out three or four hours in sun. Remembers feeling faint, giddy, and eonfused. Reeovered sufficiently in an hour to walk two miles. Returned to work in a week or ten days. Had to eease work owing to frequent faint-feelings. In Oetober, 1890, returned to England with nervousness very marked.

CASE 2.—Offieer, æt. 24. Regular habits. Four years in India. Mueh exposed to sun at musketry elass in Poona. Suddenly lost eonseiousness, and recovered only slowly. In five months was invalided for nervous shoek. Intense headaehe. Photophobia. Pain in eervieal spine. Pulse slow and irregular. Pupils dilated. Sent to England one month later and reeovered.

CASE 3.—Æt. 22. Exposure while shooting in India. Beeame insensible after returning to his quarters, with stertorous breathing and eonvulsions. Gradually regained eonsciousness, but with paresis of lower limbs, loss of speeeh, and defective intelligence. Asylum with dementia.

CASE 4.—Offieer, æt. 32. Thirteen year's serviee in India. In November, 1890, at musketry exposed to sun six hours daily without shelter. Fell uneonseious. Reeovered slowly. Mental confusion and impaired memory remained. Visit to Japan, eured; but insomnia, irritability, and giddiness recurred on return to India. One year's leave to England given.

CASE 5.—Gunner, æt. 31. Sober and steady. Admitted to hospital in India, in 1849, for sunstroke. Reeovered and left. Soon found wandering about with incoherent speech. Admitted asylum Mareh, 1851, with dementia. Died in England, 1856.

Post-mortem.—Calvaria very adherent to membranes. Skull plates unusually thiek. Diplöe obliterated. Two ozs. serous fluid in araehnoid. Pia mater opaque and thick.

CASE 6.—Offieer, æt. 43. Sunstroke in India in 1868. Followed by left headache; heavy articulation. Singing in left ear. Defective vision left eye. Memory defeetive. Conduct eeeentric. Asylum in Europe, 1870, with general paresis. Improved and resided at home for two years. Re-admitted asylum, 1874, with general paresis. Died 1879.

Post-mortem.—Calvaria dense. Diplöe obliterated. Membranes vascular, thickened, and adherent. Grey matter defieient. Convolutions flattened. Some softening of right optie thalamus.

Prophylaxis.—The head and spine should be protected from the actinic rays. This can best be done by lining the hat with a red coloured material, which is opaque to these rays.

The hat should come down well over the nape of the neck and over the temples, and it is better, if thin white clothes are worn, to sew a strip of red material down the line of the spine.

Morbid Anatomy.—Whether the ehange induced by the actinic rays, on the body tissues, is of a ehemieal or electrical nature, it is hard to say.

In the fulminating cases there may be very little obvious pathologieal condition beyond a cerebral hyperæmia.

In the chronic cases the calvaria is generally found thickened, and the membranes tough and adherent. The diplöe are almost always obliterated.

The convolutions are frequently flattened; and there may be softening of some portion of the cerebral substance.

In conclusion, it may be added that a certain number of cases are often met with, which are undoubtedly mixed cases of diathermasia and phœbism; that is to say, that exposure to the rays of a tropical sun, on the part of an unacclimatised person, will often result in a condition of phœbism (or actinic stroke) complicated by a sudden strain of the thermotaxic mechanism, resulting in a degree of fever considerably higher than that which would be met with as the reactionary fever in phœbism.

CHAPTER XIX.

DISTOMIASIS.

(Infection by distoma or flukes.)

Definition.—An infection of man, either incidental or definitive, by various trematodes (other than the *Bilharzia*).

Enumeration.—The following are the chief trematode parasites of man. Nos. 1 to 6 appear to be probably merely incidental parasites as far as man is concerned:—

1. *Gastrodiscus hominis.*
2. *Dicrocœlium lanceatum.*
3. *Cotylogonimus heterophyes.*
4. *Fasciola hepatica.*
5. *Fasciolopsis buski.*
6. *Opisthorchis noverca.*
7. *Opisthorchis sinensis.*
8. *Opisthorchis felineus.*
9. *Paragonimus westermani.*
10. *Schistosomum japonicum.*

Natural History, &c.

1. **Gastrodiscus hominis** (so named by *Lewis* and *M'Connel* in 1876).—Synonym—*Amphistomum hominis.*

It is of a reddish colour, measuring 5 to 8 mm. in length and 3 to 4 mm. in breadth. One end of the parasite opens out into a large circular disc, at the posterior border of which the small sucker is situated.

The generative organs are comprised of two testes, a vas deferens and a laterally-placed uterus, opening by a genital pore on a level with the bifurcation of the intestine.

But little is known as to the excretory or nervous systems.

The eggs are oval, 0·15 by 0·072 mm. The parasite has only been found twice (in an Assamese and in an Indian), when it was present in the cæcum and colon in large numbers. Its normal host is probably some Indian mammal. Nothing is known of its life history. It does not appear to inconvenience its human host.

2. **Dicrocœlium lanceatum** (Stil. and Hass., 1896).—Synonyms—*Fasciola lanceolata*, Schrank, 1790; *Distomum lanceolatum*, Mehlis, 1825; *Dicrocœlium lanceolatum*, Dujardin, 1845.

Body is pointed at both ends, especially anteriorly. Length, 8 to 10 mm.; breadth, 1·5 to 2·5 mm.

The oral sucker is at the anterior extremity, and measures about 0·5 mm. in diameter. The ventral sucker is distant about 2 mm., and measures 0·6 mm. in diameter.

There is a globular pharynx, connected by an œsophagus with an intestine which occupies four-fifths of the body.

The generative organs consist of testes, an ovary, and uterus; they communicate with the exterior of the body by a genital pore which opens at the level of the bifurcation of the intestine.

The ova are oval, thick-shelled, and yellow or brown in colour; they measure 0·04 by 0·025 mm. Within the ova are found oval miracidia, only the anterior part of which is ciliated. They do not hatch out in water spontaneously, but have been found to do so in the intestines of slugs, although no subsequent development has been observed in that situation.

The normal habitat of this fluke is the bile duct of domestic animals, such as the sheep, ox, goat, ass, horse, rabbit, pig, &c. It is sometimes found associated with the liver fluke, but is not as widely distributed. In addition to most European countries, it has been found in Algiers, Egypt, Siberia, and America.

In man it has only been found seven times (Germany, Italy, France, and Egypt), when it produced no special symptoms.

The intermediary host and subsequent life history are not known.

3. **Cotylogonimus heterophyes.**—Synonyms—*Distomum heterophyes*, v. Siebold, 1852; *Mesogonimus heterophyes*, Raill., 1890; *Cœnogonimus heterophyes*, Looss, 1900.

This is the smallest distome that has so far been found in man. It is 2 mm. in length by 1 mm. in breadth. It has rectangular scales with serrated posterior margins of 7 to 9 points.

The oval sucker is 0·1 and the ventral sucker 0·35 mm. in diameter.

The genital pore is close behind and to one side of the ventral sucker.

The uterus occupies a large part of the posterior portion of the body.

The ova are 0·03 by 0·017 mm., and have thick shells. Within is a miracidium ciliated on all sides, and possessing a rudimentary intestinal sac.

The species was discovered by Bilharz in Cairo in 1851. It has not often been found in man.

The usual host is the dog, more rarely the cat.

The normal habitat is the central third of the small intestine. Nothing is known of the life history of this parasite.

4. **Fasciola hepatica.**—Synonyms—*Distomum hepaticum*, Retz, 1786; *Fasciola humana*, Gmel., 1789; *Distomum caviæ*, Sons, 1890; *Cladocœlium hepaticum*, Stoss, 1892.

This very interesting and important trematode is the common "liver fluke" of a large number of herbivorous animals, and occurs in most parts of the world.

More especially in the sheep, it is highly pathogenic, and is the cause of periodical epidemics which commit great ravages—*e.g.*, in England, in 1830, over 2,000,000 sheep died from fluke disease.

The life history has been extensively studied.

In man it is only a casual parasite, and only 23 cases have been observed. The disturbance caused in most cases was only trifling, although in one or two cases there was a fatal issue preceded by enlargement and tenderness of the liver, with jaundice.

In several of the human infections the parasite was not found in its usual hepatic habitat, but in swellings or abscesses of the foot, occiput, ear, &c., whither it had probably been conveyed by the blood stream.

The parasite is a large one, being 20 to 30 mm. long by 8 to 13 mm. in breadth.

The head is about 4 mm. long, and sharply differentiated from the rest of the body.

The suckers are hemispherical and quite close to each other, the oral being 1 mm. and the ventral 1·6 mm. in diameter.

The intestine bifurcates in the head cone.

The testes occupy the greatest part of the posterior portion of the body.

The ova are oval in shape, and of a yellowish-brown colour. They have a cap-like lid, and measure 0·132 by 0·07 mm.

The clinical history of an infected sheep is as follows:—

There is first a period of invasion, occurring in the autumn, and lasting from four to thirteen weeks. The young flukes do but little harm at this period. Later, in November and December, there is a period of anæmia; the sheep fatten rapidly, become anæmic and jaundiced, and cease to eat much. The fæces are normal, but may contain fluke ova.

In January (about three months after the entry of the larvæ) a period of emaciation begins. There is irregular pyrexia and quickened respirations. There is considerable weakness, and local œdemas are common. Death generally occurs at this stage, but if they survive till May or June the flukes then emigrate from the liver, and convalescence and recovery may take place.

The *life history* was worked out by *Leuckart* and *Thomas*:—

The ova first reach water, where the enclosed miracidium, which is ciliated all over, becomes free, and it then penetrates a water snail, *Limnæus truncatulus*, which is a common occupant of large or small collections of water throughout Europe, North Africa, Afghanistan, Thibet, &c. In the absence of this snail, analogous species may act as intermediate hosts, as in the *Limnæus oahuensis* in the Sandwich Islands.

Within the body of this snail the miracidium loses all its organs, and becomes a *sporocyst*. This sporocyst is a yellowish tubular or fusiform body with a rounded edge, and contains germinal bodies. Their size increases until they give rise to *rediæ* which remain in the same host, and sometimes it develops a second generation of rediæ.

These rediæ are more cylindrical, and possess a simple intestine and a pharynx, also a genital orifice at the anterior end which serves for the exit of the cercaria originating within them. The cercaria leave their host and become encysted on the meadows, and are taken up by the definitive hosts with their food. These *cercaria* are very different from the rediæ. They have a body and an oar-like tail. They possess much the same organisation as do the adult trematodes. They have, however, additional organs, such as a boring spine and eyes, which are not found in the adult. When they become encysted they cast their tail, and the boring papilla and eyes disappear almost entirely, the genitals meanwhile becoming more developed.

5. **Fasciolopsis buski.**—Synonyms—*Distomum buski*, Lank., 1857; *Distomum crassum*, Busk, 1859.

This is the largest distome so far found in man. It may measure from 24 to 70 mm. in length, and is 5·5 to 14 mm. in breadth. The oral sucker is 0·5 mm. in diameter; the ventral sucker is quite close to it, and is about 2 mm. broad. The whole body is a somewhat elongated oval.

The genital pore is at the anterior border of the ventral sucker.

The ova are oval, and closed by a delicate operculum. They measure 0·15 by 0·075 mm.

Its normal habitat is apparently the intestine, but it is evidently only a casual human parasite. Nothing is known either of its life history or of its normal definitive host.

In man it has only been reported eight times—China, Sumatra, Straits Settlements, Assam, India, and British Guiana contributing each a case or two. In two instances the infection was associated with recurring diarrhœa, but it is apparently innocuous to the human host, as a rule.

6. **Opisthorchis noverca** (Braun).—Synonyms—*Distoma conjunctum*, Lewis, Cunningham, M'Connell, and Cobbold, 1859-72; *Opisthorchis conjunctus*, Manson.

This parasite was first found by Cobbold in the liver of an American fox (*Canis fulvus*) that died in London in 1859. It was found again by Lewis and Cunningham in the liver of Calcutta pariah dogs in 1872. Subsequently, in 1876, M'Connell found a large number of them in the bile ducts of Mohammedans who died in Calcutta.

The fluke is lancet-shaped, covered with spines and measures 9·5 to 12·5 mm. in length by 2·5 in breadth.

The two suckers are closely approximated, the oral being larger than the ventral.

The genital pore opens immediately in front of the acetabulum.

The intestinal cæca extend nearly the whole length of the fluke, from a spheroidal pharynx.

The cirrus pouch is lacking.

The ova are oval and operculated, and measure 0·035 by 0·02 mm.

It is apparently only incidentally found in man, and has but little pathological importance in that host.

Nothing is known of its life history or usual definitive host.

7. **Opisthorchis sinensis** (Cobbold, 1875).—Synonyms—*Distoma sinense*, Cobbold, 1875; *Distoma spathulatum*, Leuckart, 1876; *Distomum hepatis endemicum*, Baelz, 1883; *Distomum japonicum*, Blanchard, 1886.

The parasite inhabits the bile ducts and gall bladders of domestic dogs and cats, and also human hosts.

It was first found in a Chinaman in Calcutta in 1874, by M'Connell. Subsequently, M'Gregor found the same parasite in eight more Chinese cases in Mauritius. It was also found in a Chinaman in New York. In recent years it has been found in a few instances in Bengal Hindoos; and an enormous percentage of inhabitants in some of the provinces of Japan has been found to be infected.

The number of parasites in one host may amount to a thousand or more. They are occasionally also found in the pancreas and duodenum.

Its endemic home would therefore appear to be China and Japan, where the definitive host may be man as often as the lower animals.

The changes produced in the liver consist of local dilatations and sacculations, with proliferated connective tissue. Interstitial hepatitis is set up, and fatty degeneration of hepatic cells occurs in addition to interference with the mechanical functions by the engorgement of the bile ducts. Hepatic atrophy follows the hyperplasia, and death occurs in about 14 per cent. of the bad infections.

The parasite is yellowish-red in colour and almost transparent. It is oblong and narrow, and is pointed anteriorly.

It is 10 to 14 mm. long, by 2·5 to 4 mm. in breadth.

There are no spines. The parasite is capable of considerable contraction.

The oral sucker is larger than the ventral, and they are situated about 3 mm. apart. The genital pore is just anterior to the ventral sucker.

The ova are brownish-black, thin shelled, oval, and operculated, measuring 0·03 by 0·015 mm. (Fig. 37). They contain a completely ciliated miracidium, the subsequent development of which is not known.

8. **Opisthorchis felineus** (Rivolta, 1885).—Synonyms—*Distoma conus*, Gurlt, 1831; *Distomum lanceolatum*, v. Sieb., 1836; *Distomum tenuicolle*, Mühl., 1896.

The parasite is yellowish-red and almost transparent, much resembling in this respect *Opisthorchis sinensis*.

At the level of the ventral sucker there is a shallow constriction of the conical neck. It is considerably smaller than *O. sinensis*, and usually only measures 10 mm. by 1·5 mm.

The suckers are of equal size, and situated about 2 mm. apart from each other.

The uterus is central, and the genital pore just anterior to the ventral sucker.

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Fig. [illegible].—Ovum of *Opisthorchis sinensis* (× 500).

Fig. 58.—Ovum of *Opisthorchis felineus* (× 500).

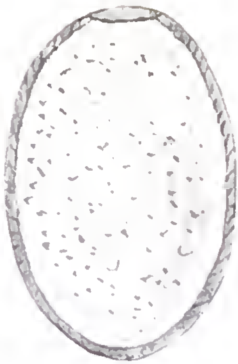

Fig. [illegible].—Ovum of *Paragonimus westermani* (× [illegible]).

Fig. [illegible].—Ovum of *Schistosomum japonicum* (× 500).

The ova are oval and operculated, and measure 0·03 by 0·01 mm. (Fig. 38).

The usual definitive host is the domestic cat, whose gall bladder and bile ducts it inhabits. It is, however, not infrequently found in the dog and the fox, and has been many times found in man. In fact, in some parts of Siberia it is the parasite most frequently met with in post-mortem examinations. Several hundred flukes may be present in one host.

It has considerable pathological importance, connected with dilatation of the bile ducts and inflammatory thickening of their walls.

Interstitial hepatitis, accompanied by some jaundice and ascites, is very often present. Subsequent atrophy of the liver seems to be the usual result, and the condition should be regarded as a grave one, although not perhaps so serious or attended with as high a mortality as is an *O. sinensis* infection.

Occasionally the fluke may also be found in the pancreas and in the intestine.

But little is known of this trematode's life history. The ova contain ciliated miracidia which do not hatch out in water. They have been found to hatch out if introduced into the intestine of *Limnæus stagnalis*, but no further development occurred.

9. **Paragonimus westermani** (Kerbert, 1878).—Synonyms —*Distoma ringeri*, Cobbold, 1880; *Distoma pulmonale*, Baelz, 1883; *Mesogonimus westermani*, Railliet, 1890.

This fluke was first discovered by *Kerbert* in a royal tiger that died in Amsterdam, being found in pairs in superficial cysts of the lungs. This was in 1878.

Soon afterwards *Baelz* observed the eggs of this species in the sputum of some Japanese who were suffering from hæmoptysis of endemic occurrence, and they were at first mistaken for the spores of gregarines. The parent of these ova was first seen in man in the bronchi of a Formosan by *Ringer*, and was described by *Manson* as *D. ringeri*, although *Baelz*, who found the ova, called them *D. pulmonale*.

It has since been observed many times in China, Japan, and Corea; and one case is supposed to have originated in Mexico.

The parasite is also found in dogs, cats, and pigs.

The fluke is reddish-brown in colour, and oval in shape.

It measures 8 to 10 mm. in length by 4 to 6 in breadth.

The suckers are of equal size (0·75 mm.). The oral sucker is subterminal, and the ventral just anterior to the middle of the body.

There are scale-like spines on the cuticle.

The excretory pore is posterior and rather subterminal. The pharynx is small and the œsophagus short.

The genital pore is eccentric, and just posterior to the ventral sucker.

The ova are thin-shelled, oval, and brownish-yellow, having an average size of 0·09 by 0·055 mm. (Fig. 39).

But little is known of the life history, except that the ova will

hatch out in water, liberating a miracidium which is ciliated all over and swims freely.

The habitat of the fluke is usually the lung, although occasionally it may reach the liver, testes, or brain. It is, therefore, probable that the eggs may be conveyed from the lung by the blood stream.

One or two flukes may be found in hard-walled cysts scattered throughout the lung. In the vicinity of these cysts the lung tissue becomes disintegrated, occasioning cavities, and sometimes opening up blood-vessels.

The symptoms consist of a chronic cough, especially urgent in the early mornings. The expectoration is rusty brown, and occasionally complicated by hæmoptysis. Many ova will be found in it.

Treatment, of course, is of slight avail. Every prophylactic precaution should be taken, however, to prevent infection when in an endemic area, by only using boiled water and exercising due care in the consumption of uncooked vegetables.

Patients should be careful to avoid spreading the infection, by paying due regard to the disposal of their sputum.

10. **Schistosomum japonicum** (Katsurada, 1904).—Synonym—*Schistosomum cattoi*, Blanchard, 1905.

The condition caused by this parasite had been noticed for some years by many observers in certain provinces in Japan, being characterised by enlargement of the liver and spleen, ascites, diarrhœa, anæmia, and occasionally pyrexia. They found numerous eggs in several organs, especially the liver. The cause of this condition was discovered by *Katsurada*, in Okayama, in 1904, who found a ciliated miracidium (not unlike those of the *S. hæmatobium*) in the ova contained in the fæces of these cases; and he also found the adult trematodes, in the portal system of two cats, having identical ova. This parasite he named *S. japonicum.*

Subsequently, in a case that died at the Quarantine Station, Singapore, of cholera, but previously had exhibited no special symptoms, *Catto* found the same ova in sections of the liver and bowel. The author, who saw these at the time, suggested a resemblance to the ova of *Bilharzia*, but they were thought by Daniels and Finlayson to be coccidia. Subsequent sections made at home disclosed the presence, in the smaller mesenteric blood-vessels, of male and female trematodes; and both the adults and ova were identified with *S. japonicum.*

Morphologically, this fluke is not unlike the well-known *S. hæmatobium* (*Bilharzia*), in that it is a trematode with independent sexes, and that the habitat of the adult is the vascular system of its definitive host. It is, however, considerably smaller.

The males measure, on an average, 10·43 mm. in length and 0·53 mm. in breadth; the females, 8 to 12 mm. in length by 0·4 in breadth. These are from *Katsurada's* measurements of fresh specimens. Those given by *Catto* are considerably less, but refer only to spirit specimens.

The integument of the male is smooth, and not spinous, as in the

e of *Bilharzia*. This would suggest an arterial habitat, in which e spines would not be necessary to prevent being carried away in blood stream, as might happen to the *Bilharzia*, which inhabits venous system.

The posterior sucker is considerably larger than the anterior.

The ova, which measure 75 μ by 40 μ (Fig. 40), are of a yellowish-wn colour, are oval in shape, and have neither spine nor oper-um. They are found chiefly in the large intestine, where rmous numbers may be seen in the submucous coat. They are plentiful in the liver and mesenteric glands, and, to a less ree, in other abdominal organs. Their presence in the tissues vokes, firstly, a small-celled infiltration, to be subsequently owed by a proliferation of fibrous tissue.

We know that in bilharziosis the infection may often cause but e inconvenience to the definitive host. From the character of ova, and their situation, it is probable that infection with *aponicum* will cause even less inconvenience still to a majority hose attacked.

Name of Trematode.	Sexual Characters.	Habitat of Adult.
1. *Gastrodiscus hominis*, . .	Hermaphrodite.	Large intestine.
2. *Dicrocœlium lanceatum*, .	Hermaphrodite.	Bile ducts.
3. *Cotylogonimus heterophyes*, .	Hermaphrodite.	Small intestine.
4. *Fasciola hepatica*, . . .	Hermaphrodite.	Liver.
5. *Fasciolopsis buski*, . . .	Hermaphrodite.	Intestine.
6. *Opisthorchis noverca*, . .	Hermaphrodite.	Bile ducts.
7. *Opisthorchis sinensis*, . .	Hermaphrodite.	Bile ducts and gall bladder.
8. *Opisthorchis felineus*, . .	Hermaphrodite.	Bile ducts and gall bladder.
9. *Paragonimus westermani*, .	Hermaphrodite.	Lung.
10. *Schistosomum japonicum*, .	Sexes distinct.	Smaller intestinal arterial system.

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Fig. 41.

1. *Gastrodiscus hominis.*
2. *Dicrocælium lanceatum.*
3. *Cotylogonimus heterophyes.*
4. *Fasciola hepatica.*
5. *Fasciolopsis buski.*
6. *Opisthorchis noverca.*
7. *Opisthorchis sinensis.*
8. *Opisthorchis felineus.*
9. *Paragonimus westermani.*
10. *Schistosomum japonicum* (male and female).

Size of Adult (in millimetres).	Character of Ova.	Size of Ova (in millimetres).	Intermediary Host.
6·5 by 3·5	Oval.	·15 by ·07	Unknown.
9 by 2	Oval. Thick. Yellow.	·04 by ·02	? Slugs.
2 by 1 Serrated scales.	Thick shells.	·03 by ·02	Unknown.
25 by 10	Oval. Brown. Operculated.	·13 by ·07	Limnæus truncatulus.
50 by 10	Oval. Operculated.	·15 by ·075	Unknown.
11 by 2·5 Spines.	Oval. Operculated.	·035 by ·02	Unknown.
Transparent. 12 by 3 Contractile.	Black. Oval. Thin. Operculated.	·03 by ·015	Unknown.
Transparent. 10 by 1·5 Contractile.	Oval. Operculated.	0·3 by ·01	? Limnæus stagnalis.
Oral sucker is subterminal. 9 by 5 Spines.	Thin. Oval. Brown.	·09 by ·05	Unknown.
Filiform. Males 10·4 by ·5 Females $\frac{5}{12}$ by ·4 Smooth.	Yellow. Oval. No spine. No operculum.	·075 by ·04	Unknown.

CHAPTER XX.

DRACONTIASIS.

(*Guinea-worm infection.*)

Definition.—An invasion of an animal host by a nematode—the *Filaria medinensis*, Velsch, 1674—contracted in certain endemic tropical areas.

Synonyms.—*Vena medinensis*, Velsch, 1674; *Dracunculus persarum*, Kämpfer, 1694; *Gordius medinensis*, Linné, 1758; *Filaria dracunculus*, Bremser, 1818; *Filaria æthiopica*, Valenciennes, 1856; *Dracunculus medinensis*, Cobbold, 1864; *Guinea worm; Medina worm.*

History and Geographical Distribution.—The recognition of this nematode undoubtedly dates from remote antiquity. It has even been thought that the "sep worm" of the Ebers Papyrus (B.C. 1550), refers to this parasite.

The first positive statements are found in Plutarch (B.C. 150) who states that people on the Red Sea littoral had a disease resembling the exit of small snakes from the skin. The Greek name for the worm was δρακόντιον, from which is derived the Latin designation of *dracunculcus*, and the modern term for the condition, dracontiasis.

Galen promulgated the idea that the condition was not due to a parasite, but partook more of the nature of varicose veins, and this was largely the current opinion until comparatively recent times, although Velsch, Lind, and others had demonstrated its parasitic nature during the seventeenth and eighteenth centuries.

Its endemic distribution is confined to comparatively limited areas of the affected countries, and would appear sometimes to die out, as has been the case in British Guiana and the neighbourhood.

The following are the regions in which guinea worm is to be found:—The West Coast of Africa, Nubia, the Soudan, the Arabian Red Sea littoral, the coasts of the Persian Gulf and of the Caspian Sea, Turkestan, the Western Coast of India from Bombay to Kutch, Rajputana, the Deccan, and, according to Scheube, in Fiji.

Natural History and Etiology.—The females (Fig. 42) attain a length of 50 to 80 cm. or more, and average 0·5 to 1·7 mm. in breadth. They are of a yellowish colour. The cuticle is firm and elastic, and white or yellow in colour. The anterior end is roundish. The mouth has two lips, behind which are two lateral and four sub-median papillæ. At the posterior end is a spine, 1·0 mm. in length, and ventrally directed. Below the œsophagus the

[To face p. 204.

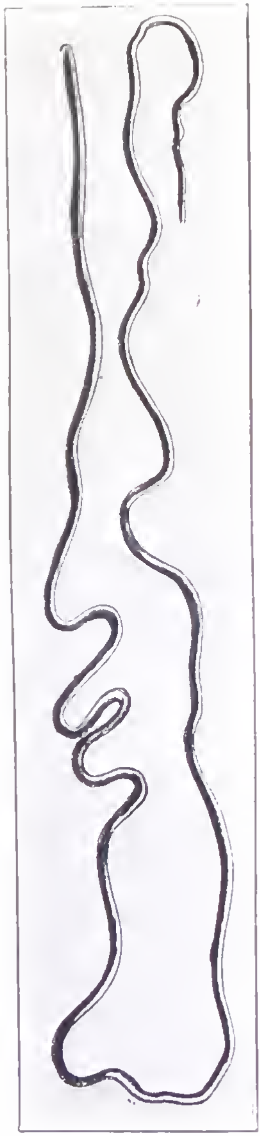


Fig. 4.—Female guinea-worm (*F. medinensis*), after *Leuckart*.

alimentary canal is atrophied. The vulva and vagina are not known. Most of the body is occupied by the uterus. In 1902 the male worm was discovered by *Charles*, who when making a post-mortem in Lahore found two female worms in the subperitoneal tissue; and clinging to each was a smaller worm, 4 cm. in length, attached by its posterior extremity to a point about 14 centimetres from the head of the female. It is assumed that these males were attached to the vulva of the females, as in the case of *Syngamus trachealis*, and that they die after copulation.

Habits.—The adult lies in the connective tissue, between the muscles or under the skin.

When mature, with marvellous intuition, she generally moves down to a leg or foot, never wounding blood-vessels or nerves and rarely opening a joint. Having arrived at her destination, and ovulation being completed, she drills a little hole in the derma, over which a small blister is formed in the epidermis. By and by this blister bursts or is ruptured, disclosing the small ulcer leading to the worm, or from which a small portion of head may be seen protruding. Sometimes, at this stage, some absorption of toxines elaborated by the parasite may cause a little rise of temperature or urticarial eruption in her human host.

The worm is at this stage in a dilemma. The contractility of her uterus is abolished by the enormous distension with her embryos; the same distension has obliterated the vagina; and finally, in order to live, her millions of embryos must in some way or other reach water. At first sight nature would seem to be defeated. But in reality it is quite the reverse, as an experiment will show. If some cold water be allowed to drip on the leg or foot in the neighbourhood of the little ulcer, in a few seconds some milky fluid will exude, and after ten or more seconds the flow will cease. Under the microscope this milky fluid will be found to consist of countless embryos, which if placed in water will become active and live for eight or ten days. A further application of water will not again provoke this parturition until several hours have elapsed.

This process may be repeated daily until one day part of the worm itself will be found extruded. If now the douching process is repeated we shall at last see how it is that the embryos could escape although the vagina was obliterated. Careful observation will show the projection of a beautiful delicate and pellucid tubule from the worm's mouth, to the extent of about three-quarters of an inch. The contents of this tubule are at first clear, but gradually become milky, as material is forced into it from behind. It now becomes tense, ruptures, discharges the milky fluid and collapses. The collapsed portion shrivels up and closes the womb. On re-application of the stimulus some hours later, a fresh portion of the womb is extruded, and ruptures in like manner; and so on till the entire uterus is expelled and parturition at an end. Her life's object being thus attained the worm dies, and may either be absorbed, expelled, or removed by artificial traction.

This marvellous effort of nature sounds like some wonderful fairy-tale, but it is capable of demonstration at any time.

It has been already remarked that the guinea worm usually presents itself in a leg or foot. In fact, in natives, this happens nine times out of ten. In acting thus, the worm shows a prescience which is almost uncanny. The first step in the extra-corporeal life of the embryo must be in water.

The part of the native's body which comes into most frequent contact with water is the foot or leg. By some instinct the worm seems to know this and makes therefore towards these parts. With the bheesties or water carriers of India, whose dripping skins are slung over their shoulders, the guinea worm will frequently present itself on the back.

With the bath-loving European, the parasite will frequently present itself on the scrotum, thighs, or trunk.

It is thus almost certain that by some means the worm becomes cognisant of the parts most frequently in contact with water.

Incidence.—No race, age, or sex is exempt.

The infection is usually single, but occasionally two, three, or even ten worms may be present.

Diagnosis and Prognosis.—The symptoms are usually only those of a localised boil-like inflammation of the skin. The wanderings of the worm seldom cause any sensation, though there may be an occasional feeling of heaviness, burning, itching, or dragging; this may increase till there is actual pain and inflammatory swelling of some consequence. The prognosis is, as a rule, quite favourable.

Course.—After the preliminary infection, the life development of the worm occupies about one year.

Observations seem to point to an especially favourable condition for the human infection, occurring at a certain time each year, in the endemic area. Seasonable influences, such as the rains, may perhaps contribute to this result; but it is certain that the incidence rate is especially high during one month in the year, in certain places, such as the Gold Coast (*Graham*).

Treatment.—Until parturition is completed, the worm resists extraction. If, therefore, traction is attempted before this the consequences may be disastrous. If the worm snaps, the escape of the uterine contents may result in violent inflammation, abscesses, and sloughing. Perhaps even serious contractions; or death from septic trouble might result.

The proper treatment, therefore, is to aid in parturition by cold douches for the fifteen or twenty days until it is completed. The worm may then be slowly wound out.

A French naval surgeon, *Emily*, injects the body of the worm, when protruding, with a solution of perchloride of mercury, 1 in 1,000. This kills the parasite, and extraction after twenty-four hours is easy.

Life History of the Embryos (Fig. 43).—The larvæ, if

[illegible] *p.* 156.

Fig. [illegible] Guinea-worm embryos. (Drawn from a photo by the author.)

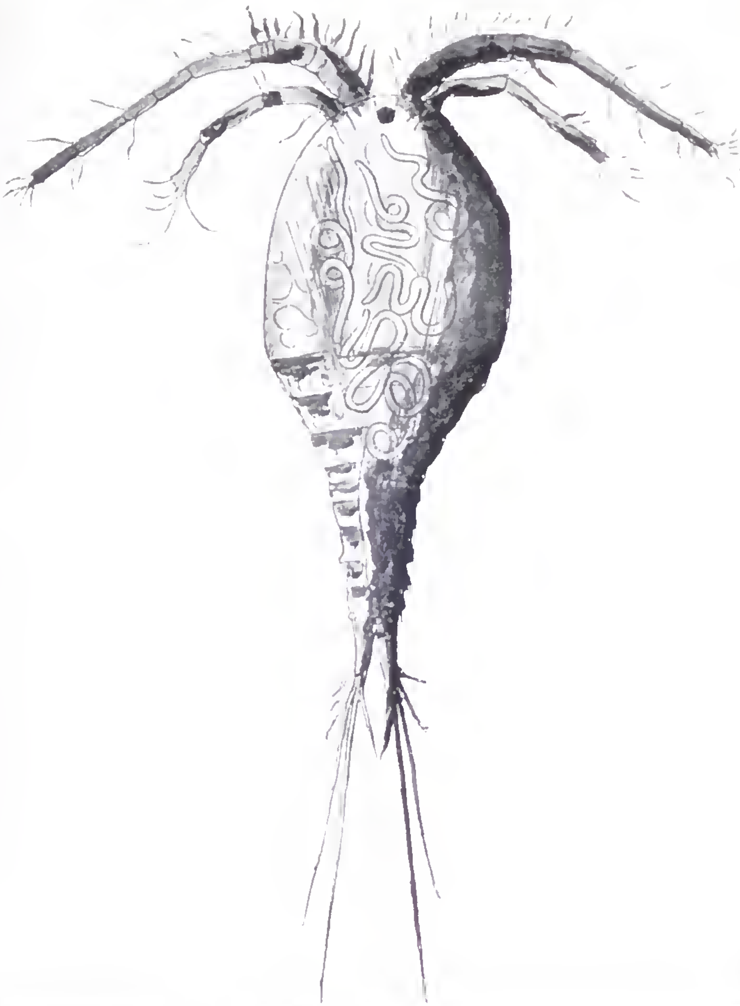


Fig. 41. Cyclops quadricornis after *Leuckart*

examined, when extruded, exhibit scarcely any movements; but, if placed in water, become highly active, and will live for some days.

In consequence of their resemblance to the embryos of *Cucullanus elegans* (a parasite of *Perca fluviatilis*), which live in a small crustacean—the cyclops, *Fedschenko*, in Turkestan, on the advice of *Leuckart*, introduced some larvæ into water containing cyclops, and was able to demonstrate their invasion of the wee crustacean. This was confirmed later by *Manson*.

The host used was the fresh-water cyclops (*C. quadricornis*, Fig. 44).

On about the twelfth day the embryos cast their skin, and assumed another form. They could be observed within the cyclops until the fourth week; yet no changes occurred though the larvæ grew to a length of 1 millimetre.

The experiment of feeding cats and dogs on water or milk in which infected cyclops were present was tried with negative results.

Recent work by *Leiper* has thrown some light on the subject.

An infected cyclops, at the fourth week, in which the embryos have become quiescent, if placed in some 0·2 per cent. hydrochloric acid (representing the acidity of the gastric juice) will be gradually killed, "showing little motion beyond a few tremors of the antennæ and rami. On the other hand, as the acid penetrates, the apparently quiescent filaria regains its former activity. At first slowly, but with every moment with increasing strength and rapidity, it coils, stretches, and re-coils, until at last, having divested itself of what probably is the remains of a very delicate cuticle, it bursts into the general body cavity of the now moribund cyclops, and in a very short time completely destroys the organised contents by its increasingly violent motions. The larva is soon seen to be testing with obvious purpose every joint of the cuticular integument. At one moment it is attempting to advance along the entire length of a narrow antenna, at the next it may be found attacking one or other of the prolongations of the bifurcated tail. Retreating again it will endeavour to ascertain the patency of the anus, the genital openings, or the mouth. By one or other of these apertures, or, in some cases, it may be by a rupture of the cuticle, the young worm eventually reaches the exterior, and there swims about with great rapidity, like a minute snake.

"One specimen, watched for three hours, continued with brief rests, in which it coiled and uncoiled, to manifest the same marvellous vitality."

Other like experiments made with the cyclops at the sixth, instead of the fourth week of infection, showed the embryos to be, by then, dead.

It seems probable, therefore, that the larvæ, with their intermediate hosts, are introduced into the stomach of human beings in drinking water, and there become free. Here they may possibly attain puberty and copulate; the male dying off and passing out with the fæces, and the female beginning to travel towards the subcutaneous tissues.

The possibility of the entry of the embryos through the human skin has been considered, but no proof adduced.

CHAPTER XXI.

DYSENTERY.

Definition.—Dysentery is an inflammation of the large intestine, leading to thickening and ulceration of the mucous and submucous coats; being characterised by the passage of frequent and scanty stools containing blood and mucus, accompanied by tormina and tenesmus, and frequently by fever.

History and Geographical Distribution.—Dysentery, probably confounded often with diarrhœa, typhoid, and other intestinal fluxes, has been mentioned and known since remote periods.

It is thought that Eber's papyrus (B.C. 1550) has references to the disease.

Hippocrates, about 400 B.C., describes the condition, as also *Galen* in the second century of the Christian era. The latter attributed it to the action of the bile—an opinion held for about fourteen subsequent centuries.

The gross anatomical changes were first described by *Morgagni* in 1747.

The first tropical reports of the disease were by *Bontius* from the East Indies in 1642, and by *Piso* from the West Indies in 1648.

Dysentery occurs in all latitudes. Sporadic and epidemic dysentery may be met with anywhere, and are quite frequently seen in temperate or even cold climates.

Endemic dysentery, however, is limited to tropical and subtropical countries only. The following are the principal centres of the disease. The African Continent, Syria, Arabia, India, the Malay Peninsula and Archipelago, China, Japan, the Pacific Islands, and a large part of South America.

Etiology.—In most accounts of dysentery, the treatment of its etiology is either nebulous or involved, and frequently amounts to a discussion on the relative merits of an amœba or a bacillus as the chief etiological factor.

Moreover, it is often overlooked that there may not only be several varieties of bacilli associated with dysentery, but also several varieties of amœba.

The most convenient etiological classification is as follows:—

1. Sporadic.
2. Endemic (or amœbic).
3. Epidemic (or bacillary).

	Sporadic.	Endemic.	Epidemic.
…stribution,	May occur anywhere.	Limited to the tropics and sub-tropics alone.	May occur anywhere.
…usal agent,	1. Mechanical irritation, or 2. *Balantidium coli* (*Paramœcium c.*), or 3. *Bacillus coli communis*, or 4. Other causes.	*Entamœba Dysenteriæ* (*E. histolytica*).	1. Shiga-Kruse bacillus, or 2. Lenz bacillus, or 3. Flexner-Strong bacillus, or 4. Harris-Gay bacillus.
…sociated …rganisms,	*Bacillus coli communis.*	*Entamœba coli.* *Paramœba hominis.* *S. pyogenes aureus.*	*Bacillus coli communis.*
…urse, .	Mild acute disease.	Tends to be chronic. Amœbæ usually found in stools.	Is an acute disease, less severe in the varieties 2 to 4 (paradysentery bacilli), than if the infection is due to the dysentery bacillus No. 1.
…lapses .	Unusual.	Common. One attack predisposes to a recurrence.	Rare. Immunity conferred by severe attack.
…e, . .	May occur at any age.	Rare in old age and in infancy.	May occur at any age.
…ver abscess,	Never occurs.	Found as a complication or sequela in from 5 to 50 per cent. of cases, according to country of origin or nationality of patients.	Never occurs.
…ugs, . .	Ipecacuanha no good. Purgatives indicated.	Ipecacuanha has marked effect.	Ipecacuanha no use. Intestinal antiseptics indicated.
…ethod of …nfection, .	Impure food and water.	Impure water.	Impure water. Wind-borne dust. Fly-borne infection. Personal uncleanliness. Latrine origin.

I. **Sporadic Dysentery.**—The following causes of sporadic dysentery have been recorded:—

(*a*) *Mechanical Irritation.*—In the S. African War *Faichnie* records clinical symptoms of dysentery traceable to carrots issued in Maconachie rations. Want of mastication resulted in a dysenteric condition, and slices of carrots were passed in the stools.

(*b*) *Balantidium* (*Paramœcium*) *coli.*—This is one of the ciliated infusoria and is normally an intestinal parasite of swine. It differs morphologically from the usual paramæcia of water. *Klein* isolated it from several of the London waters, and considered it as pathognomonic of sewage pollution. *Strong* and *Musgrave* report a fatal dysentery due to this cause.*

(*c*) *Bacillus coli communis.*—In a considerable number of cases of sporadic dysentery the B.C.C. is the only organism to be found. *Celli* and *Fiocca* consider that this bacillus may acquire under certain circumstances very virulent properties.

That this would seem to be the case is indicated by an example in the author's practice, in which the rice-water stools of a typical cholera case showed an almost pure culture of the Colon bacillus.

(*d*) *Bacillus pyocyaneus.*—*Calmette* and *Maggiora* have found this bacillus to be apparently the cause of certain cases of dysentery; and several cases in the United States have been ascribed to the same cause by writers in the *Journal of Experimental Pathology*, 1898.

(*e*) *Durham's Micrococcus.*—*Durham* isolated a very minute micrococcus from the blood, liver, bile, &c., of seven cases of asylum dysentery. This, however, is of questionable etiological importance (*Hewlett*).

II. **Endemic Dysentery.**—This is also called "Tropical dysentery" or "Amœbic dysentery."

Scheube has adduced two facts to invalidate an amœbic origin of dysentery. In the first place, he says that there are cases of dysentery in which no amœbæ are present. This, of course, is no proof that amœbæ never cause dysentery, but merely points to a probable bacillary origin in those cases. He then says that amœbæ are frequently found in the stools of healthy people who have never had dysentery. This also, of course, is no argument, since it is a well-established fact that diphtheria bacilli may be found in the throats of healthy people who are not suffering from diphtheria, and that both cholera and typhoid bacilli may occur in the stools or bowels of healthy people who show no signs of those diseases.

There are three species of amœbæ found in man:—

1. *Entamœba dysenteriæ* (Schaudinn).
2. *Entamœba coli* (Schaudinn).
3. *Paramœba hominis* (Craig).

Only the first of these is pathogenic and is the cause of tropical or endemic dysentery.

* *Johns Hopkins Hosp. Bull.*, xii., 1901.

The other two are non-pathogenic; their presence is adventitious, and they will not produce dysentery in cats.

The following are the chief differential points in these three amœbæ:—

	E. dysenteriæ.	*E. coli.*	*P. hominis.*
Size, . .	25 to 35 μ.	10 to 15 μ.	15 to 20 μ.
Cysts, . .	No cysts.	Encysted forms occur.	Encysted forms occur.
Colour, . .	Often greenish.	Opaque grey.	Opaque grey.
Pseudopodia,	*Ectosarc* highly refractile and glassy, and stains intensely. *Endosarc* coarsely granular, and stains less intensely than ectosarc.	*Ectosarc* less refractive. *Endosarc* finely granular, and stains more intensely than ectosarc.	Protoplasm is homogeneous.
Vacuoles, .	Contains a mass of vacuoles and many red cells.	Generally no vacuole and only one or two red corpuscles.	No vacuole.
Nucleus, .	Lateral. Hard to see in fresh specimens. About 5 μ in diameter.	Central. Distinct. Encapsuled. Highly refractile. Proportionally large.	Eccentric. Spherical. Small. Replaced later by refractive dots.
Mobility, .	Active.	Limited.	Limited.
Reproduction,	Division, and formation of spores at periphery of endosarc.	Division, and formation of cysts with eight daughter amœbæ.	Undergoes flagellate and amœboid development, like the water amœba *P. gilhardi* (Schaudinn).

III. **Epidemic Dysentery.**—Called also "Bacillary dysentery." This acute, non-liver-abscess-forming disease is due, in the majority of cases, to *B. dysenteriæ* (*Shiga* and *Kruse*).

In addition to this, there are three other forms allied to the parent stock (called *para-dysentery bacilli*), which are capable of producing

the same morbid condition, though usually rather less acutely. In some epidemics one or other of these para-dysentery bacilli may be the causative factor instead of the Shiga bacillus.

These para-bacilli are:—The *Lenz* bacillus, the *Flexner-Strong* bacillus, the *Harris-Gay* bacillus.

The tabular cultural features (after *Shiga*), on p. 203, will serve to differentiate them.

The following extract from the *Conclusions of the Royal Commission on S. African Dysentery, 1900*, will not be out of place, referring as they do to the epidemic (bacillary) type so prevalent during the Boer War.

(*a*) The organs in dysentery are sterile. It is a local disease attacking the mucous and sub-mucous coats of the large intestine, and, unlike enteric fever, the causal agent, if any, confines itself to the intestines.

(*b*) There is no connection between dysentery and enteric, Eberth's bacillus being found neither in the organs nor intestines of dysentery cases.

(*c*) There is a certain amount of evidence to show that so-called cases of dysentery following enteric fever are relapses of enteric, where the disease has attacked the large intestine.

Symptoms and Treatment.—There is a great variety in the character and urgency of the symptoms in cases of dysentery. The essential symptoms of all kinds of dysentery are those of a colitis.

1. Frequent muco-sanguineous stools.
2. Griping.
3. Tenesmus.

The latter is most urgent if the lesions are near the rectum: if nearer the cæcum, then the griping will be the most prominent feature.

I. **Sporadic Dysentery** is usually associated with a preliminary diarrhœa, which develops into the usual dysenteric manifestations after a few days. By degrees the dysenteric element entirely supersedes the diarrhœa. The griping and tenesmus ensure an almost constant attendance at the commode. There is scarcely any fever; but the tongue is dirty, and there is anorexia.

The condition is usually amenable to treatment within three or four days, and does not tend to become chronic.

The **treatment** should consist of the preliminary exhibition of a dose of castor oil, followed by—

℞—Mag. sulph., ... ʒss.
Aq. cinnamon, ... ℥i.

M. F. Mist.

Sig.—℥i. every hour till free purgation is produced. Discontinue if the stools become watery.

TYPES OF DYSENTERY BACILLI.

(Slender rods; 1 to 3 μ in length.)

	Shiga-Kruse.	Lenz.	Flexner-Strong.	Harris-Gay.
Indol, . . .	–	±	+	+
Dextrose, . .	+	+	+	+
Mannite, . .	–	+	+	+
Saccharose, . .	–	–	+	+
Maltose, . .	–	–	–	+
Dextrin, . .	–	–	–	+
Lactose, . .	–	–	–	–
Acid formation, .	–		+	
Liquefaction of gelatin, . .	–	–	–	–
Gram, . .	–	–	–	–
Mobility, . .	–	–	–	–

The diet should consist only of milk, or milk and soda, and rest in bed should be enjoined until there has been cessation of the mucoid stools for at least three days. The return to normal diet should be very gradual. Alcohol is not, as a rule, necessary until convalescence is established.

II. **Amœbic Dysentery.**—For a day or two before the attack there may be a preliminary diarrhœa ; the stools gradually become mucoid ; streaks of blood are soon seen ; tenesmus becomes urgent, and soon the typical dysenteric condition is set up. There may be a little irregular fever, but this is generally not a constant symptom. The stools may average from twenty to sixty, or more, per diem. The condition, after continuing for four or five days (during which there has been a very large secretion of mucus), then becomes altered. The stools become a thin reddish liquid, consisting of blood, pus, debris, and mucus. Examination will show the presence of numerous amœbæ, and Charcot-Leyden's crystals are sometimes found. These acute symptoms may either—

1. Gradually terminate in convalescence.
2. Proceed to aggravated ulceration or gangrene.
3. Become sub-acute, with relapses.
4. Become chronic.

1. In the first case the symptoms slowly abate.

2. In the second case there is no abatement of the symptoms. The number of calls to stool will increase until the patient is unable to leave the commode. The stools become highly offensive. Sloughs are passed. There is great weakness. The pulse is small and fast. The skin is cold and moist. This state is highly critical and dangerous, and not infrequently terminates in death from exhaustion during the second week of the disease. A typhoid state is often set up. In the most favourable circumstances the condition will modify somewhat, and gradually pass into a relapsing state of chronic dysentery.

3. In a certain number of cases the initial acute symptoms do not entirely disappear. The stools resume something of their normal character, but are rather loose, and accompanied still by griping. After a week or two a chill or a slight error in diet will cause a relapse, and the active symptoms will recommence.

Partial recovery is continually followed by relapses, until at last the patient may die of exhaustion, or a slow convalescence may be established. Liver abscess is a not infrequent sequela.

4. The condition of chronic amœbic dysentery is usually a sequela of a primary aggravated ulceration, as stated in class No. 2 above. The condition may persist for years, and entails much misery, pain, and emaciation.

The **treatment** of these cases of **amœbic dysentery** should be as follows :—

(*a*) *If acute* :—

1. Absolute rest in bed.
2. Milk (3 pints daily).
3. Brandy as required.
4. Ipecacuanha.

    No food for three hours.
    Tr. opii, ♏ xx. in water.
    Mustard plaster to epigastrium. In half an hour give

    ℞.—Ipecac. pulv., ... gr. xxx.

5. Repeat the ipecac. treatment twice daily for a day or two until the urgent symptoms abate.
6. Enemata of 0·2 per cent. solution of eucalyptus gum.
7. Hot fomentations to abdomen.

(*b*) *If chronic* :—

1. Rest.
2. Milk diet.
3. Ipecac. for four evenings in diminishing doses.
4. The following mixture :—

℞.—Tr. simarubæ, ...	℥ss.
Decoct. hæmatoxylin,	
Decoct. granati cortic, āā, . . .	℥ij.
Aq. cinnamon ad	℥i.

M. Ft. Mist.

Sig.—℥i. every three hours.

5. The following enemata, given twice daily :—

℞.—Eucalyptol, ...	1·5
Eucalypti gummi, ...	2·5
Aquæ ad	1500

M. Ft. enema.

Or else :—

℞.—Quin. sulph., ...	15
Acid. tannic., ...	30
Sod. chlorid., ...	6·5
Aq. dest. ad	1500

M. Ft. enema.

6. During convalescence :—No cold baths. No alcoholic drinks. Warm clothing. Simple and digestible food. Sea voyages. Tonics.

III. Bacillary Dysentery.—The *incidence* of the attack is generally much *more abrupt* than in the case of amœbic dysentery. For some hours—perhaps for a day—the stools may be loose and fæculent. The tongue is foul, the appetite lost. Abdominal pains

become marked. Urgent calls to stool result in the passage of mucoid dirty material soon streaked with blood.

The *temperature usually rises several degrees*, rigors being not uncommonly met with (Figs. 45 and 46).

Tenesmus and dysuria are painful and prominent features. These acute symptoms may either—

1. Fulminate ;
2. Proceed to aggravated ulceration ; or
3. Terminate in convalescence.

In these bacillary dysenteries *relapse* or *chronicity is rare*.

1. The fulminating cases show a fairly high temperature throughout. Rigor, vomiting, and headache are usual accompaniments. The usual dysenteric symptoms are, as a rule, very urgent. In three to four days the temperature will drop to subnormal, and collapse cause the death of the patient. The mortality in this type of disease is exceedingly high.

Occasionally it may happen that the bacillary toxæmia is so great that death occurs before pronounced dysenteric symptoms have declared themselves.

2. Cases proceeding to aggravated ulceration are frequent in some epidemics. The symptoms are much like those occurring in the same class of amœbic cases (*q.v.*), except that they rarely develop into a chronic condition, and the passage of massive sloughs is less frequent.

3. Cases of slight severity proceeding to convalescence have no special features which call for remark.

The **treatment of bacillary dysentery** should be as follows:—

(*a*) *If acute:*—

1. Absolute rest in bed.
2. Milk diet (3 pints daily).
3. Alcohol, if required.
4. Salines:—

℞.—Sod. sulphatis, ... gr. lx.
Acid. sulph. arom., ... ♏xx.
Aq. cinnamon ad . . . . . ℥i.

M. Ft. Mixt.

Sig.—℥i. every two hours till relief is obtained.

5. Enemata of pure olive oil.
6. Hot fomentations to abdomen.
7. Antidysenteric serum (made from the proper type).

(*b*) *If sub-acute:*—

1. Rest and milk.
2. Antidysenteric serum.
3. Intestinal antiseptics:—

℞.—Salol, ... gr. xv.

Every three hours.

EPIDEMIC (BACILLARY) DYSENTERY.

(Charts from the *S. African Royal Commission Report.*)

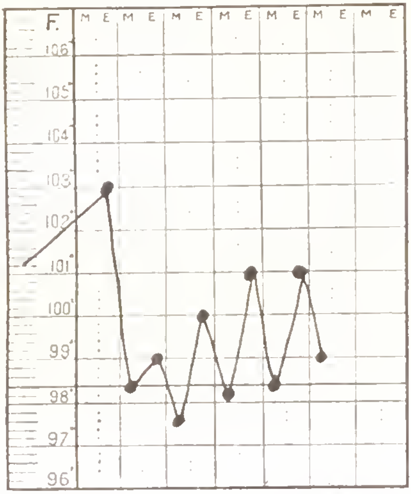


Fig. 45.—European soldier. Aged 22. Death. (Large intestine showed pre-ulcerative inflammation. No amœbæ. Mucous membrane invaded with bacilli.)

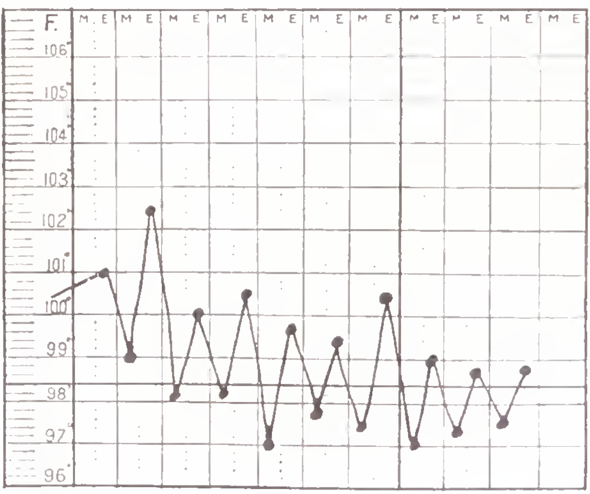


Fig. 46.—European soldier. Aged 32. Death. (Advanced ulceration of large intestine. No amœbæ. Bacteria plentiful.)

4. Hot fomentations to abdomen.
5. Enemata of a 0·1 per cent. solution of ol. gaultheria.
6. During convalescence:—Warm clothing. Digestible food. Tonics.

General Hints.—The dejecta should be mixed with formalin.

The bed-linen should be frequently changed and disinfected.

It is very grateful if the body is frequently sponged with a solution of eau-de-Cologne.

The anal aching, tenesmus, and constant desire to go to stool may be somewhat checked by the approximation of the patient's legs and the application of a hot bottle to the back of his thighs, with one end close to the perineum.

Diagnosis and Prognosis.—Taking the standpoint of the definition as given in this chapter, the diagnosis of the condition should present no difficulties.

To differentiate, however, between an amœbic and bacillary origin is not always easy. The following factors will aid in a diagnosis:—

Amœbic.	Bacillary.
There is known to be a tropical endemic focus; and liver abscesses are reported.	May occur anywhere, either in tropics or temperate zones. Usually epidemic. Not associated with liver abscess.
Does not occur in epidemic form in villages or amongst bodies of men.	Usually occurs in epidemic form.
Amœbæ are found in the stools.	Bacilli may be found in the stools.
Fever is unusual.	Fever is frequent.
Onset often gradual.	Onset usually sudden.
Frequently tends to become chronic.	Is usually an acute disease.
Yields as a rule to ipecacuanha.	Ipecacuanha has no effect.
Blood does not agglutinate cultures of dysentery bacilli.	Blood gives reaction with dysentery bacilli.

It should be borne in mind, however, that cases of a mixed infection are met with occasionally.

The *prognosis* will depend partly on the constitution and age of the patient, and partly on the type of the disease.

If properly treated and not allowed to go on to a chronic condition, the prognosis in amœbic cases is fairly good. If it becomes chronic, it is less good; and the possibility of liver-abscess should be considered.

Bacillary dysentery is always a more severe disease. Fulminating cases may occur. The toxæmia consequent on the bacillary invasion of the gut produces a febrile state, and largely adds to the dangers of the condition.

In all cases the larger the area of ulceration the greater the danger of hæmorrhage, perforation, and septicæmia.

Morbid Anatomy.—The intestinal changes vary with the intensity of the inflammatory process.

In mild cases the summits of the valvulæ conniventes are covered with a greyish-white layer of mucoid debris, under which the mucous membrane is rather eroded in parts. Elsewhere it is hyperæmic and softened. There is also some infiltration of the submucous tissues, and the solitary glands are large and prominent.

In more severe cases there is greater implication of the mucous membrane and submucous. The intestinal wall is thickened in patches. The enlarged solitary glands usually slough, giving rise to ulcers. The muscular and serous coats are implicated. The gut is much dilated.

In the most severe cases there is very extensive necrosis, a large area of the mucous membrane being converted into black sloughs.

In chronic conditions, the submucous changes become more marked. New fibroid growth gives rise to considerable induration and permanent thickening, causing contraction and narrowing of the lumen. Occasionally abscesses and fistulæ are seen in the thickened wall.

The *liver* is usually hyperæmic and enlarged in cases of acute dysentery. In 4 per cent. of cases of amœbic dysentery, the liver is the seat of single or multiple abscesses (see Chapter XXV.).

In chronic cases the liver may be atrophied, and frequently shows fatty degeneration.

The *mesenteric glands* are usually enlarged and congested in acute cases; in chronic cases they are fibrous and pale.

The *other viscera* are not usually affected. *Perforation* may result in peritonitis.

CHAPTER XXII.

FILARIASIS.

Definition.—An infection of man, mammals, birds, &c., by various species of the order *Nematoda*, family *Filaridæ*, genus *Filaria*.

History and Geographical Distribution.—In the ninth and tenth centuries Elephantiasis was well described by Arabian physicians under the name of *Dâ-alfil*—Elephantine disease.

Endemic hæmato-chyluria was described by *Chapotin* in 1812 as occurring in Mauritius. Nothing further was added to the knowledge of this subject until *Demarquay*, in Paris, in 1863, discovered an embryo nematode in the chylous hydrocele fluid from a Havanese.

Wücherer, in 1866, found the same kind of embryos in chylous urine of some patients in Brazil.

Two years later (1868), and without knowledge of the previous discoveries, *Lewis* observed the same parasites in Calcutta in the urine of a case of chyluria, and shortly afterwards in the blood of a patient suffering from chronic diarrhœa. Having thus found the blood to be the habitat of this embryonic filaria, he named it *Filaria sanguinis hominis*. Since that time the subject has received much attention.

Manson's work in Amoy demonstrated the filarial periodicity of the blood embryo, which he called *F. nocturna*. He also pointed out that this was not the only blood worm of man. In blood from patients coming from Old Calabar and the Congo he found a somewhat similar nematode, but with diurnal periodicity; this he named *F. diurna*. In blood of other natives over a wide region of West Africa was found another blood embryo differing very much from the others and exhibiting no periodicity. This Manson named *F. perstans*.

In blood sent by *Newsam* from St. Vincent, West Indies, was another different filaria, called by Manson *F. demarquaii*. This same parasite has also been found by *Low* in St. Lucia, Dominica, and Trinidad.

In native blood sent by *Ozzard* from British Guiana embryos were discovered with different peculiarities again. These Manson has named *F. ozzardi*.

More recently still *Magalhäes* found two adult filariæ in the left cardiac ventricle of a child who died at Rio de Janeiro (*F. magalhäesi*).

Prout has found a very large embryo in the peripheral circulation of a native in Sierra Leone (*F. gigas*).

In 1906 *Ashburn* and *Craig* discovered yet another species of filarial embryo in the blood of a prisoner at Manila (*F. philippinensis*). Besides this short historical account of the actual parasite, it will be of interest to note that observations on the further development of filarial embryos in mosquitoes were published by *Manson* and *Bancroft*, demonstrating the rôle of the mosquito as an intermediate host. *Low*, in sections prepared from material sent by Bancroft of Australia, showed that the filaria found their way into the proboscis, an observation noted also by *Dutton* in W. Africa and *James* in India.

Etiology.—Many filariæ find their habitat in human beings, other mammals, birds, &c. The filariæ of mammals will be briefly enumerated here, but are of merely general scientific interest, since they rarely, if ever, come within the domain of human medicine.

One other important filaria (*F. medinensis*, syn. *Dracunculus medinensis* or guinea-worm) is of sufficiently peculiar habit and individual importance to form the subject of a special article (see Chapter xx.).

The following tabular statement will show the names of the adult filaria at present known to frequent the human form, together with the names given to their embryos circulating in the peripheral blood.

As far as our knowledge extends, none of these have any pathological significance except *F. bancrofti*. A glance at the "site of predilection" in the accompanying table will sufficiently explain the reason for what would otherwise seem an anomaly. The situation in which *F. magalhäesi* was found will suggest that this filaria also may probably have a serious pathological significance.

Characters of the Genus and Species.

(After *Stephens* and *Christophers*, &c.)

Filariæ are long, slender worms of almost uniform breadth. The anterior extremity is rounded, and the mouth often has no lips. The males are smaller than the females. They have an incurved or spiral tail, sometimes with a thickened cuticle.

They usually have four pre-anal and a variable number of post-anal papillæ and spicules.

In the females the vulva opens near the mouth.

Of human filariæ, man is the definitive host, and certain species of culicidæ the intermediary hosts.

Of other mammalian or avian filariæ, fleas, ticks, and lice may possibly be intermediary hosts as well as mosquitoes, since filarial embryos have been found to develop in these insects, &c.

F. loa.—This parasite is peculiar to W. Africa, and is non-pathogenic. The male measures 25 to 30 mm. in length by 0·3

HUMAN FILARIÆ.—CHARACTERISTICS OF ADULTS.

Parental forms,	*F. loa*	*F. perstans*	*F. ozzardi*	*F. demarquaii*	*F. magalhäesi*	*F. bancrofti*
Name of embryo,	*F. diurna.*	*F. perstans.*	*F. ozzardi.*	*F. demarquaii.*	Unknown.	*F. nocturna.*
Pathogenicity, .	Nil.	Nil.	Nil.	Nil.	Probable.	Very marked.
Endemic area, .	W. Africa.	W. Africa. —— Demerara.	British Guiana.	W. Indies.	Brazil.	Most of tropics and subtropics.
Site of predilection of parental forms, . .	Connective tissues.	Post abdominal and thoracic connective tissues.	Sub-peritoneal connective tissues.	Omental tissues.	Left ventricle of heart.	Lymphatic trunks and varices.
Length, . .	M. 25-30 mm. F. 30-40 mm.	M. 45 mm. F. 70-80 mm.	M. 80 mm. F. 85-150 mm.	M. (unknown). F. 85 mm.	M. 83 mm. F. 155 mm.	M. 80 mm. F. 85-150 mm.
Thickness, . .	M. 0·3 mm. F. 0·57 mm.	M. 0·12 mm. F. 0·25 mm.	M. 0·21 mm. F. 0·3 mm.	M. ... F. 0·32 mm.	M. 0·4 mm. F. 0·7 mm.	M. 0·2 mm. F. 0·26 mm.
Head to Vaginal outlet, Ovarian opening	...	0·6 mm. ?	0·71 mm. 0·85 mm.	...	...	0·71 mm. 0·92 mm.
Tail to anal papilla, . .	...	0·145 mm.	0·230 mm.	...	...	0·225 mm.
Nature of tail, .	Incurved, with two terminal tubercles.	Bulbous, incurved, and mitred.	Bulbous. —— Cuticle not thickened.	...	...	Blunt, circular, not bulbous.

HUMAN FILARIÆ.—CHARACTERISTICS OF EMBRYOS.

Name.	Length.	Breadth.	Sheath.	Head.	Tail.	Anterior v. Spot.	Viscus.	Posterior v. Spot.	Motility.	Periodicity.
F. diurna, . . (*F. loa*)	0·3 mm.	0·0075 mm	Present — Loose	6 lips	Pointed	Present	Granular mass	Present	Lashing — Not progressive	Diurnal
F. perstans, . . (*F. perstans*)	0·2 mm.	0·0045 mm	Absent	Papillated	Blunt	Negative	Negative	Negative	Lashing and progressive	Nil
F. ozzardi, . . (*F. ozzardi*)	0·21 mm.	0·005 mm	Absent	?	Pointed	?	?	?	Progressive ?	Nil
F. demarquaii, . (*F. demarquaii*)	0·2 mm.	0·005 mm	Absent	Spine	Pointed	Present	?	?	Progressive	Nil
Embryo not known (*F. magalhaesi*)	..	..	..	..	..	..	..	..	..	..
F. philippinensis, (?)	0·32 mm.	0·0005 mm	Present — Tight	Serrated retractile band	Pointed and abruptly attenuated	Present	A spiral tubular cylinder	Present — Also a papilla	Lashing and progressive	Nil
F. nocturna, . (*F. bancrofti*)	0·3 mm.	0·0075 mm	Present — Loose	6 lips	Pointed	Present	Granular mass	Present	Lashing — Not progressive	Nocturnal

mm. in breadth. The female, 30 to 40 mm. in length by 0·5 mm. in breadth.

The tail of the male is incurved, and has five large papillæ on each side of the anus, and two short unequal spicules. The mouth is simple and punctiform, without armature.

Its habitat is the connective tissue, which it traverses freely. It is not infrequently obvious when crossing the sub-conjunctiva or tissues about the orbit, and the male apparently will sometimes chase the female. It is probably long-lived, as it has been found in a person who had left the endemic area for some ten years.

Prout, *Armett*, *Dutton*, and *Elliott* have come across loa infection in which *F. diurna* was found in the blood. This blood filaria is therefore thought to be the embryonic offspring of *F. loa*. These *F. diurna* much resemble *F. nocturna*. They possess a loose sheath. Length 0·3 mm., breadth 0·0075 mm. The head has six lips. The tail is pointed. The motion is lashing, but not progressive, and there is a diurnal periodicity in its appearance in the peripheral blood. If the sleeping habits of the human host are inverted, and he sleeps during the day instead of the night, there is *no* corresponding inversion of the periodicity of *F. diurna* (as happens in the case of *F. nocturna* under similar experiment).

F. perstans.—This parasite is fairly common in West and Central Africa, and has been reported from Demerara. It is non-pathogenic.

The parental form was discovered by *Daniels* in Demerara Indians, in the connective tissues at the root of the mesentery, behind the abdominal aorta, and in the sub-pericardial tissues.

The male is smaller than the female, being 45 mm. long by 0·12 mm. broad, as compared with the female length of 70 to 80 mm. and breadth of 0·25 mm.

The tail is bulbous, incurved, and has two triangular appendages resembling a mitre.

The embryos which inhabit the peripheral blood show no periodicity. They are 0·2 mm. long by 0·0045 mm. broad. They are thus considerably smaller than *F. diurna* or *F. nocturna*. They have no sheath and the tail is quite blunt. With the exception of *F. gigas*, this is the only blunt pointed embryo as yet known.

F. ozzardi.—The parental form was found by *Daniels* in the sub-peritoneal connective tissues of the anterior abdominal wall of a Demerara Indian.

The male is 80 mm. by 0·21 mm. and the female 85 to 150 mm. by 0·3 mm. They are non-pathogenic.

Their embryos (*F. ozzardi*) are 0·21 mm. long by 0·005 mm. broad. They are found in the peripheral blood, and exhibit no periodicity. They have no sheath; the tail is pointed.

F. demarquaii.—A parental female filaria was found by *Galgey* in the omental tissues of a patient in the West Indies, in whose blood *F. demarquaii* embryos had been present. This was probably the parent. No male was found.

The female measured 85 mm. by 0·32 mm. They are not pathogenic.

It is found in St. Vincent, St. Lucia, Dominica, and Trinidad.

A somewhat similar embryo has been found in New Guinea, so that a wider endemic area possibly may exist; but its distribution is strangely limited even in the endemic districts.

The embryo (*F. demarquaii*) is 0·2 mm. long by 0·005 mm. broad. It exhibits no periodicity, has no sheath, and possesses a pointed tail.

F. magalhäesi.—Professor Magalhäes found two adult filariæ (male and female), now called *F. magalhäesi*, in the left ventricle of a child's heart, that died in Rio de Janeiro.

The male measured 83 mm. in length by 0·4 mm. in diameter. The female was 155 mm. in length by 0·7 mm. in diameter.

The mouth was circular and unarmed. The male tail had four pairs of pre-anal, and four pairs of post-anal papillæ, and two spicules.

No blood examination had been made during life. The nature of its embryos is unknown, nor have we any knowledge of its associated pathology or life history.

? **F. gigas.**—The parental form of this is unknown. It is apparently not pathogenic.

The embryos were described by *Prout* as occurring in the blood of a native at Sierra Leone. No description of measurements is to hand, but they are very large, non-sheathed, and blunt-tailed, and have a strong staining affinity for fuchsine.

It has been said later (*Low*) that the filaria found by Prout was in reality not a filaria at all, but an accidental contamination by hairs from the leg of a fly.

F. philippinensis. The adult form is not known, but is apparently not pathogenic.

The embryos were found in 1906 by *Ashburn* and *Craig* in the blood of a Visayan prisoner in Bilibid prison, Manila.

The length is 0·32 mm., and the breadth 0·0065 mm. It is thus rather longer, though less slender, than *F. nocturna* or *F. diurna*.

It exhibits no periodicity. There is a tightly fitting sheath, never distended or flat as with *F. nocturna* or *F. diurna*, but extending beyond the ends of the body as a fine thread resembling a flagellum.

The anterior V spot is about 0·105 mm. from the margin of the head. There is a viscus in the central third of the body in the shape of a convoluted or spiral tube.

A posterior V spot and papilla can be made out in fresh specimens, but not in stained.

Midway between the posterior V spot and the tip of the tail there is a sudden attenuation, from which offset there is a progressive diminution to a very fine point.

Its motion is lashing and progressive. It does not appear in large

numbers in the peripheral blood, the greatest number found in one slide being seven.

The next filaria (*F. bancrofti*), being pathogenic to human beings, will be dealt with at greater length.

Filaria bancrofti.—The embryo of this parasite (*F. nocturna*, Fig. 47) was first found by *Demarquay* in Paris, in 1863, in some chylous fluid from a hydrocele. Again, in 1866, by *Wucherer* in some chylous urine. Still later, in 1872, in the blood of a patient in Calcutta, by *Lewis*.

Since that time it has been found widely distributed in almost every tropical and subtropical country, from Spain to W. Africa, and from the United States to China, and the Pacific Islands and Australia.

The parental form, named by *Cobbold F. bancrofti*, after Bancroft of Brisbane, who was the first to find it (1876), has been found very many times. The sexes are usually found in association, perhaps up to six or seven in number, all coiled up together, and lying in the lymphatics of the trunk or extremities. When first exposed they wriggle somewhat, and have the exact appearance of a short length of white horse-hair. The male is 80 mm. long by 0·2 mm. broad, and the female 85 to 150 mm. by 0·26 mm. The uterus is double; the alimentary canal is simple and straight. The young filariæ (*F. nocturna*) are ejected into the lymph surrounding the parent, and are carried to the blood stream through the lymphatics and the thoracic duct. In the blood stream they exhibit a curious periodicity. At about 6 or 7 o'clock in the evening these small embryos begin to appear in the peripheral blood, increasing in numbers up to midnight, and gradually decreasing thereafter until, at 8 or 9 a.m., no more are to be found. *During the day they retire to the lungs and the larger blood-vessels.* This was proved by the post mortem on an infected native in London, who one day committed suicide just after the filarial retiring time. No embryos were found in his peripheral blood, nor in the liver or spleen, and very few in the kidneys or brain, but in the lungs and aortic and cardiac blood thousands were found in every drop. No suitable explanation—mechanical, chemical, or vital—has yet been offered to account for this periodicity, which evidently must be in the interests of the parasite. If sleep is taken during the day and the patient keep awake at night, then the periodicity is inverted and the embryos will be found only during the day.

It is estimated that in an average sized man some 40 to 50 millions of these embryos may be circulating; but notwithstanding the incessant activity and enormous numbers of these blood filariæ, they do no harm whatever, and the human host will be as unconscious of them as of his own blood-corpuscles.

An abnormal **condition of the blood** is produced, however. *Gulland* has shown that there is a distinct *eosinophilia* in these cases. The percentage of eosinophiles is normal in the morning when no filariæ are to be found, and rises steadily up to a maximum

Fig. 17   Filaria nocturna

percentage of about 8 towards midnight, when the parasites are most numerous. This eosinophilia has been remarked in various other kinds of helminthiasis, and apparently the function of the eosinophiles is to protect the body from the toxines of parasites.

Subsequent History of the Embryo.—It is obvious that the embryo cannot of itself leave its human host. Therefore, in order to reach another person it is necessary that it shall be in some way abstracted. That it circulates in the peripheral blood at night naturally suggests that some nocturnal biting insect is the means by which it is removed. Such a blood-sucker would, of course, need to have a co-extensive geographical range. The external agent answering to all these requirements was thought, and subsequently proved, to be a mosquito.

Mosquito Hosts.—The following have been proved to act as hosts (*Stephens* and *Christophers*):—

Culex—
- *C. pipiens.*
- *C. ciliaris.*
- *C. fatigans.*

Anopheles—
- *P. costalis.*
- *Myzomyia rossii.*
- *Myzorhynchus sinensis.*

(*Note.*—*F. immitis* of the dog has been proved capable of development in *Anopheles claviger.*)

Developmental Stages of the Filarial Embryo in the Mosquito.—About an hour after the mosquito has gorged the infected blood, the blood becomes viscid. The filariæ, finding their movements impeded, commence violent movements inside their sheaths, which are now more or less fixed. The short spine and hooked lips at the head of the embryo soon succeed in piercing the sheath, and the embryo is now free. Some die, but many now attack and bore through the wall of the mosquito's stomach, and will probably reach the muscles in twelve to eighteen hours. Here movement ceases, and the body becomes thicker. Gradually a mouth becomes indicated, and, by the time the embryo is 0·3 mm. long, an anus has appeared in front of the tail, and a mouth is very distinct with four fleshy lips.

Lengthening takes place, and, at the seventh day, the cuticle will be found to be cast and some anal papillæ formed, while the parasite has attained a length of 1·5 mm.

Movements become active, and the filariæ gradually work their way through the muscular tissues till they reach the neighbourhood of the mosquito's salivary glands and pass into the neck, where one or more can readily enter the substance of the labium. Most

of the labium is chitinous, but *Dutton* has shown the point of exit to be a very delicate membrane closing in the extreme end.

When this mosquito next feeds on man, the filaria can thus gain a fresh human host. It ultimately finds its way to the lymphatic trunks, where union of the sexes may occur, and the swarm of embryos is eventually poured into the circulation, thus completing the endogenous and exogenous cycle.

Technique of Blood Examination.

1. Make fairly thick blood slides.
2. Fix with alcohol.
3. Treat till decoloured with acetic acid (·2 per cent.).
4. Wash, and stain with carbol-fuchsine.

Technique of Mosquito Sections.

1. Preserve in glycerine.
2. One day in 5 per cent. acetic acid.
3. One day in aqueous formalin (50 per cent.).
4. One day in absolute alcohol.
5. One day in absolute alcohol and ether (P. æ.).
6. One day in celloidin.
7. Section, and place two hours in strong hæmatoxylin.
8. Decolourise in 1 per cent. HCl and alcohol.
9. Wash, xylol, and mount.

Pathological Results of Parental Filariæ in Lymphatics, &c.

1. Lymphangitis.
2. Varicose glands.
3. Lymphatic varices.
4. Chyluria.
5. Elephantiasis.
6. Chylous ascites and chylous diarrhœa.

1. **LYMPHANGITIS.**—Although in many cases of filarial infection, even with *F. nocturna*, no injurious influence may be manifested, yet in a certain proportion of cases obstructed lymphatics will produce prejudicial effects.

In these cases lymphangitis is a common occurrence. The painful cord-like swelling of the lymphatics and their glands are usually early apparent. The connective tissue and skin become inflamed and tense, and an accompanying rigor and fever set in. *Fayrer* has termed this "elephantoid fever," since it habitually occurs at varying intervals in almost all cases of elephantiasis.

The swelling will subside gradually, leaving some thickened integuments.

The *treatment* should consist of rest and elevation, with local anodynes.

2. **VARICOSE GLANDS.**—Varicose groin glands are very common. The author has seen about 0·1 per cent. of such cases amongst 1,200,000 Chinese coolies arriving at Singapore. The condition is sometimes associated with a chylous œdema of the tunica vaginalis, and sometimes with chyluria.

As a rule, the condition is painless and insidious, and may involve one or both groins. The skin is freely movable over the glands, but the glands are not, as a rule, movable over the subjacent fascia. They should not be mistaken for hernia, since they are not lymphatic on percussion, nor is there impulse on coughing. Such chronic swellings in persons from a known endemic area should always be regarded as suspicious.

In treatment, they are best left alone. Surgical anastomosis between a dilated lymphatic and a neighbouring vein has been tried by *Godlee* with some success.

Varicosity of glands in other situations is more rare.

3. **LYMPHATIC VARICES.**—Sometimes superficial, and it may be transient, varices are met with on the abdomen, legs, arms, or elsewhere, probably due to the actual presence of parental filariæ.

Lymphangiectasis of the spermatic cord is not uncommon (*Manson*).

Lymph-scrotum is a term applied to a variable number of lymphatic varices in the scrotal wall. If punctured, they discharge a milky chyle. They are quite common in filarial disease. Usually embryos can be discovered in the lymph.

Frictional irritation often gives rise to elephantoid fever. The ultimate result is the formation of small abscesses, and a permanent thickening of the scrotum until a state of elephantiasis is reached.

The treatment should be directed towards suspension, protection, and cleanliness. If, however, there be frequent inflammation, or a state of elephantiasis be commencing, the removal of diseased tissues may be tried, and the wound will probably heal well. The interference with the varix may cause chyluria or elephantiasis of the leg, and the patient should be so informed.

4. **CHYLURIA.**—Filarial obstruction in the thoracic duct may cause a lymphatic varix in the bladder walls. If this ruptures, the contents will escape into the urine and chyluria result.

Usually there are some prodromata, such as pain or aching in the back or pelvis, but very often the onset of the condition is sudden.

Sometimes chylous coagula will cause retention, and thus draw attention to the condition. Frequently relapses come and go at irregular intervals. Predisposing causes of the condition may be—Frequent child-birth, sudden violent exercise, &c. Anæmia and depression are produced, though the condition is not directly fatal.

If the chylous urine be allowed to stand, the milky fluid separates into three layers. The upper layer is a cream-like pellicle, consisting of fatty matter and oil globules. In the centre there is a

thick stratum in which floats a pinkish globular clot ; it contains much granular fatty matter. The lower layer is a scanty reddish sediment, consisting of red blood cells, lymphocytes, granular fatty matter, epithelial cells, and urinary salts. In a considerable portion of cases, though not all, embryos will be found in this sediment.

The *treatment* should consist of bed, elevation of pelvis, careful diet, laxatives, and absolute rest.

Many drugs have been recommended, such as gallic acid, benzoic acid, glycerine, tr. ferri perchlor., quinine, salicylate of soda, thymol, and methylene blue, but none of them have any specific effect.

If occurring in certain places, such as Egypt, Mauritius, or Madagascar, where bilharziosis is endemic, the possibility of that disease should be considered in all cases of hæmato-chyluria, and the microscope brought into diagnostic requisition.

5. **ELEPHANTIASIS.**—*Manson* states that this is by far the most frequent manifestation of filarial invasion, although in the author's observations on over a million and a half of Chinese coolies, only two cases of elephantiasis were seen, as against over 1,500 lymph-scrotum and varicose groin glands.

The lower extremities are most commonly affected (95 per cent.). The scrotum, arms, mammæ, vulvæ, &c., are more rarely attacked.

The skin is rough and coarse, the hair sparse, the nails thick and deformed. There is no distinct line of demarcation between diseased and healthy skin. The integuments are brawny and do not pit much.

The *onset* commences with a lymphangitis, cellulitis, and an attack of elephantoid fever.

The acute symptoms subside, leaving a permanent inflammatory thickening of the integuments.

Acute exacerbations at longer or shorter intervals continue to increase the condition of elephantiasis, which may assume enormous dimensions. Where inflammatory conditions have been present, the lymphatic œdema is never permanently recovered from; but in early non-inflammatory cases recovery may be secured by pressure and elevation.

The *treatment* of the various elephantoid conditions is as follows :—

Of the Legs.—Elastic bandages ; massage ; elevation ; excision of longitudinal skin strips ; care.

Of the Scrotum.—When unsightly or inconvenient—remove by operation.

Of the Arms.—Massage ; elastic bandaging.

Of the Vulva or Mammæ.—If inconveniently large—remove by operation.

Chylous ascites or diarrhœa are very rare filarial manifestations, and should be treated on general principles.

Prophylaxis.—1. Anti-mosquito campaigns. 2. Isolation of the infected. 3. Mosquito nets for all, in the endemic area.

List of Mammalia Filariæ (after *Stephens* and *Christophers*) —1. *F. immitis.*—In right ventricle of dog, fox, and wolf. Embryos in blood. Abstracted by Anopheles. Develop in their Malpighian tubes.

2. *F. recondita.*—Female adult found in kidney of dog. Embryos will develop in Ct. canis (dog flea) and P. irritans; also in a dog tick. Transmission not yet proved.

3. *F. equina.*—Serous cavities, intestines, and liver of horses, donkeys, and mules. Embryos in blood.

4. *F. hæmorrhagica.*—Tissues of horses and donkeys. Form skin tumours.

5. *F. irritans.*—"Summer sores" of horses and donkeys.

6. *F. evansi.*—Lung and mesentery of camel. Embryos in blood.

7. *F. lachrymalis.*—Eyes of horses and cattle.

8. *F. osleri.*—Adults cause broncho-pneumonia in dogs.

CHAPTER XXIII.

GRANULOMA ENDEMICA.

Definition.—A specific granuloma of the skin, endemic in certain limited tropical areas, characterised by a very indolent ulceration which is inoculable, and due to a specific parasite identical with that of Kala-azar.

Synonyms.—*Oriental sore* (Tilbury Fox); *Biskra*, *Aleppo*, *Bagdad*, or *Delhi boil; Sahara chancre; Yearly boil* (Arabian, Turkish, and Russian); *Mycosis cutis chronica* (Carter); *Lupus endemicus* (Lewis and Cunningham).

History and Geographical Distribution.—The first accounts of Granuloma endemica we find in the middle of the eighteenth century, given by *Pococke*, *Russell*, *Volney*, and others, who reported on its occurrence in Syria.

From time to time various authors, such as *Brocq*, *Nicolle*, and others, have found various cocci which were thought to be the cause of the disease. *Fleming* and *Smith* found parasites and their ova; and *Carter* found fungi—all of which proved to be adventitious.

Not until 1885, however, did *Cunningham* (and later, in 1901, *Firth*) describe certain minute bodies in the basal exudate of the lesion. They regarded these as parasitic, and Firth alludes to them as *Sporozoa furunculosa*. Staining had not then reached the excellence since obtained, and it was not until 1904 that the American observer, *Wright*, again brought the specific bodies into prominence; and, by Romanowsky staining, showed them to be similar to the parasites found in the spleen of cases of Kala-azar, and in the bullæ of Pemphigus contagiosus. These parasites we now call Leishman-Donovan bodies. (For fuller description see Chapter xxvi.)

The disease range of distribution is a wide one, and extends over many tropical and sub-tropical countries. The following are some of its endemic areas:—Morocco, the Sahara, Egypt, Crete, Cyprus, the Crimea, Aleppo (in Syria), Bagdad (in Mesopotamia), Yemen (in Arabia), Persia, the Caucasus, Taschkent (Turkestan), Lahore and Moultan (Punjab), the Rajputana States, and Delhi (N.W. Provinces).

It has also been reported by Juliano in Brazil, at Bahia.

In the above areas it is by no means widespread, but is limited to certain towns and districts. It is more common in cities than in the country, and usually most prevalent at the beginning of the cool season.

Etiology.—The following points should be borne in mind :—

1. It is due to a specific parasite, the *Leishmania donovani* (Leishman-Donovan bodies), which is found in the tissues surrounding the ulcer in every case of Granuloma endemica.
2. The disease sticks to an endemic area.
3. It is more common in towns than in the country.
4. Neither race, age, nor sex play a part in the etiology, though perhaps it is more often found in childhood.
5. The majority of case lesions occur on the uncovered parts of the body, such as the hands, fore-arms, or face.
6. It is a chronic condition, lasting for many months, or even a year, before healing.
7. It is contagious and auto-inoculable.
8. One attack confers immunity.
9. The incubation period, as determined by inoculation, varies from three to twelve days.
10. Although due to the same parasite, Granuloma endemica is apparently never followed by such constitutional infection as Kala-azar.
11. The normal channel of transmission, and by what intermediate host, have, so far, not been discovered.

Symptoms.—As a rule, there is a primary itching papule. After a few days the papule becomes covered with dry white scales, which later become moister and adherent, forming a crust. The crust is scratched off, and discloses a shallow ulcer. The ulcer has a congested periphery, an irregular surface, and a sharp-cut jagged edge ; it discharges a thin fluid from its surface, and the area gradually advances from the periphery. Discoloured, indolent granulations form at the base of the ulcer, and frequently break down.

The *number* of boils on a patient varies. There may be only one or two, but sometimes ten, twenty, or more : and these occasionally become confluent.

Weber has observed 43 boils on one person. Out of Weber's 83 cases, in 87 the lower limbs were affected ; in 73 the arms : in 17 the face ; and in 6 the trunk.

There is usually troublesome *itching*, but *pain* is rare. Disturbances of the *general health* never occur. In some cases the primary nodule does not ulcerate, but attains the size of a bean, and disappears after a few months (Deperet and Boinet's "*forme abortive*"). The *cicatrix* left is more or less sunken, is often pigmented, and contraction frequently causes disfigurement.

Pathological Histology.—Researches have been undertaken by Carter, Riehl, Leloir, Unna, and Kuhn, who all found it to be a *chronic sero-fibrinous inflammation.* There is a round-celled infiltration of the skin and subcutaneous tissue. In the centre of the nodules this infiltration is so dense that the tissue elements are entirely disintegrated. The lymphatic vessels are dilated, and there is much œdema.

Necrosis proceeds in the centre of the infiltrated tissue. There is cornification of the hair root-sheaths, preventing a subsequent new growth of hair at the spot. There is endothelial proliferation of the blood-vessels, often leading to their entire obliteration.

The exemplification of the parasites by Wright has been already mentioned.

Diagnosis.—Syphilis, lupus, scrofula, and leprosy must be excluded, but this should present no difficulty. The place in which the patient lives and the finding of the parasites in scrapings taken from the ulcer should preclude mistakes.

Prognosis.—This is, as a rule, favourable, except for the disfigurements. Death rarely results, although it may occasionally occur from intercurrent affections, especially erysipelas.

The Jews of Bagdad were wont to anticipate the probable infection of their children by inoculating them themselves on some covered part of the body, thereby avoiding unsightly disfigurements.

Prophylaxis.—Injuries to the epidermis should be carefully avoided, also the bites of insects or the ravages of blood-sucking flies. This, together with great personal cleanliness, should prevent the possibility of attack.

Treatment.—In view of the nature of the disease, complete and early excision of the primary papule seems to be the only rational treatment.

Emily advises boracic acid applications.

Duncan recommends bandaging a thin piece of sheet lead over the ulcer, the reason for which is scarcely obvious.

If the patient is anæmic tonics are indicated; and change of climate, if the disease is of exceptionally long duration.

CHAPTER XXIV.

GRANULOMA VENEREA.

)efinition.—An infectious granuloma occurring in warm climates, .nd usually confined to the genitals and contiguous parts.

Synonyms.—*Ulcerating granuloma of the pudenda; chronic 'enereal sores; perforating granuloma of the thigh.*

Geographical Distribution.—The first communication was rom Conyers and Daniels in 1896, who observed the condition in iegroes and East Indians in *British Guiana;* Manson has seen it n *South China;* Macleod and Maitland in *India;* Goldsmith in *Vorth Australia;* Daniels in *Fiji.*

It is thus probably widespread in the tropics, though hitherto but ittle noticed.

Etiology.—The *virus of the disease is unknown,* but is indoubtedly contained in the granuloma exudation.

It has never been found before the age of twelve or thirteen, and arely after fifty. Both sexes and all races are liable to attack, especially women.

Experience has proved that it is undoubtedly *contagious,* and, noreover, *auto-inoculable,* as the extension by contiguity shows.

Its site of predilection warrants the conclusion that it is a disease of *venereal character,* and cases of its spread by sexual intercourse iave been reported.

Histological investigations have been made by Galloway on material supplied by Daniels. In the corium and papillæ there s an early small-celled infiltration; the epithelium becomes thin and itrophied; the vessels of the cutis are dilated; connective tissue akes the place of cells, but there is no tendency to caseation nor ire any giant-cells to be found.

Symptoms.—The disease usually begins on the genitals, or ieighbouring parts, as a somewhat small and delicate nodule, the epithelium of which is easily broken down, and leaves an ulcerated surface, usually superficial in extent. From this ulcerated focus a œtid, sanguineous fluid exudes, and the disease tends to spread gradually, either by contiguity or continuity.

At the edges, and in the hairy parts, the granulations are larger han at the centre.

Occasionally, when the secretion is scanty, scabs or cicatricial issue may form in the granulations, only, alas, to break down in a short time; in fact, spontaneous cure has, so far, not been observed.

Its *situation* is usually at first either on the penis, labiæ, or

thighs; it then spreads either down the thighs, or involves the scrotum, vagina, perineum, &c. Maitland describes two cases in which the lesion occurred on the mucous membranes of the cheek, gums, lips, and tongue.

Cicatrisation may occasionally set up strictures of rectum or urethra, but, as a rule, only the superficial structures are involved.

The *course of the disease* is very slow, and may extend over many years. There is no pain, however; the lymph glands are not involved, and the general health is almost unaffected.

Very prolonged duration will, of course, cause anæmia, and the patient may succumb to exhaustion.

Diagnosis.—Its large area, granular appearance, chronic course, absence of cachexia, non-amenability to mercury, &c., should differentiate it from syphilis.

Its pudendal situation, its mucous, as well as cutaneous partiality, the absence of the tubercle bacillus, of caseation, of lymph-gland participation, should preclude the diagnosis of lupus, or other tubercular origin.

Granuloma endemica and yaws might also be thought of, but the differentiation should present no difficulties.

Treatment.—*Conyers* and *Daniels* recommend the local application of

℞.—Acid. salicyl., ...	2
Ung. creasoti., ...	30

M. Ft. ung.

Scraping, and the thermo-cautery, may be used with indifferent success. Complete and early excision offers the best chance of a permanent cure.

Maitland recommends frequent applications of Vienna paste, followed by boracic acid poultices.

Of internal drugs—mercury and potassium iodide have proved of no value.

The administration of calcium iodide might possibly meet with the admirable success which attends its exhibition in many chronic ulcerative conditions:—

℞.—Calc. iod., ... gr. ij.

Ft. cachet.

Sig.—t. d. s.

CHAPTER XXV.

HEPATITIS AND LIVER ABSCESS.

Hepatitis.

'HE liver is, of course, subject to the various affections, both ınctional and organic, to which it is liable in cold climates, as ımours, cirrhosis, lardaceous or fatty degenerations, calculi, &c.

Beyond and above all these, however, there is a special predisosition to liver affections caused by residence in tropical climates. 'his is perhaps due mainly to the following causes :—

1. High temperature at first stimulates the liver and increases ecretion ; it then diminishes the activity and prevents the secreting ower (Fig. 48).

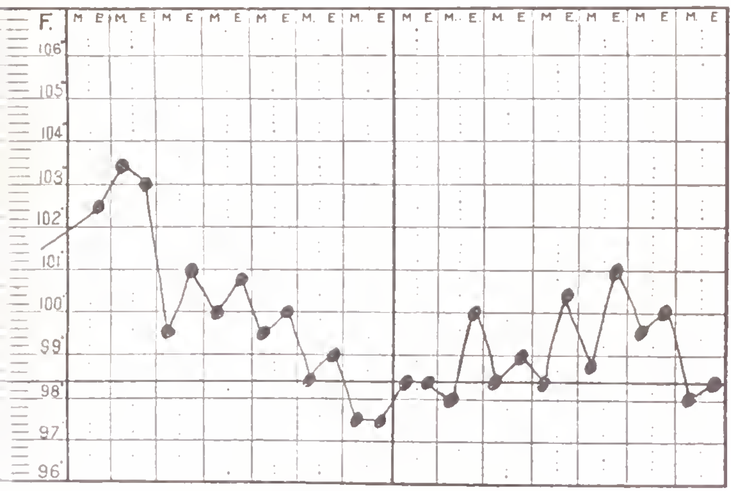


Fig. 48.—Hepatic congestion—European male, aged 34 years.
(*From the Author's Case Book.*)

2. There is less atmospheric oxygen in hot climates, leading to ssened activity of the lungs and diminished CO_2 output, and conequently entailing more work for the liver.

3. Tropical residence tends to general impairment of nutrition ıd deterioration of blood, in which the liver, as an excretory organ, ıares.

4. The resident in warm climates is too apt to maintain the same food standard to which he has been accustomed in cold climates, a proceeding to which his liver is likely to take exception.

5. The unusual diaphoresis is apt to produce unusual thirst, too often resulting in dangerous alcoholic indulgence, and consequent hepatic congestion.

6. Many tropical diseases (but especially dysentery and malaria) have an important connection with the liver.

These and other causes throw an unusual amount of work on the liver, causing first hyperæmia and then congestion. This early stimulation results in increased secretion and elimination of bile. In the new-comer this will generally mean—headache, dirty tongue, heavy sleep, attack of fever, bilious diarrhœa, and dull hepatic aching.

The years go on, too often in the paths of physiological unrighteousness, and attacks, such as the above, do not convey the warning intended by nature.

A sudden chill, indiscretions in diet, exposure to the sun, &c., may at any time convert such a congested liver into an *acute hepatitis*—Rigors, pyrexia, anorexia, constipation, pain and tenderness over liver, enlargement of hepatic dulness, pain in right shoulder and down arm, hurried respirations, weakness and depression, tongue furred, jaundice, and clay-coloured stools are the result.

Or, on the other hand, the attacks of hyperæmia and congestion may not lead to a definite acute hepatitis, but rather to a gradual diminution of liver activity leading to a perverted power of secretion. A condition is then attained such as is very often noticed in Anglo-Indians—Pale sallow complexion, listlessness, constipation or irregular bowels, bad digestion, pasty motions, irritable temper, chronic enlargement of liver.

Such cases, if still in the tropics, are liable to attacks of acute hepatitis, and are bad subjects to contact malaria or dysentery.

If they retire to a cold climate, it is important to avoid chill, or alcoholic indulgence.

The **treatment** of these cases is important.

I. **The early febrile attack of hyperæmia and congestion.**

(*a*) The patient should keep quiet in the house.

(*b*) The following purge should be administered:—

R.—Extr. colocynth co., ...	gr. iss.
Hyd. subchlor., ...	gr. i.
Extr. jalap, ...	gr. i.
Pulv. gambog, ...	gr. ¼

M. Ft. pil.

Sig.—Pil. i. statim.

(*c*) A mustard plaster should be applied over the hepatic region.

(*d*) The following effervescing mixture should be administered for two or three days:—

Ṛ.—Sod. bicarb., ... ʒiij.
Ammon. carb., ... ʒss.
Aq. ad . . . . ℥vj.

Ṛ.—Acid. cit., ... ʒiss.
Syr. limon., ... ʒvj.
Aq. ad . . . . ℥vj.

Sig.—℥i. of each to be mixed and drunk during effervescence one hour before each meal.

(*e*) The diet for a few days should consist only of fish, eggs, beef-tea, and milk.

(*f*) In subjects who have once had an attack, fulness or aching in the liver region may indicate the possibility of another attack. This should be warded off by rest, care in diet, and a dose, such as—

Ṛ.—Sod. sulph., ... ʒi.
Sod. bicarb.,
Sod. chlorid. āā., ... gr. xxx.
Aq. ad . . . . ℥i.
M. Ft. mist.

Sig.—℥i. at bedtime for two nights.

II. An Acute Hepatitis.

(*a*) Rest in bed.
(*b*) Milk diet.
(*c*) Ṛ.—Hyd. subchlor., ... gr. v.

To be followed after some hours by—

Ṛ.—Mag. sulph., ... ʒi.
Acid. sulph. arom., ... ♏xx.
Aq. ad . . . . ℥i.
M. Ft. haust.

(*d*) A hot bath, followed by bed and blankets, and a large hot poultice over the whole liver, the latter being repeated twice daily.

(*e*) The following mixture should be administered for some days:—

Ṛ.—Ammon. chlor., ... gr. xx.
Extr. cascar. sagrad. liq., . . . ♏xxx.
Aq. ad . . . . ℥i.

Sig.—℥i., t.d.s.

(*f*) When the pain and temperature have gone and the hepatic dulness is reduced, the poultice may be replaced by a flannel binder and the mixture discontinued. Other

bland or slop foods may now be given in addition to the milk.

(*g*) As convalescence is established it is well to give a hepatic tonic, of which the following is a good example:—

℞.—Acid. nitro-hydrochlor. dil., . . .	♏viij.
Tinct. nucis vom., ...	♏v.
Extr. taraxaci liq., ...	♏xxx.
Aq. chlorof. ad	℥i.

M. Ft. mist.

Sig.—℥i., t.d.s.

Tropical Liver Abscess.

Definition.—An abscess formation in the liver, either single or multiple, occurring chiefly in male Europeans, connected with residence in the tropics or subtropics, and associated with amœbic dysentery.

(*Note.*—This chapter does not deal with those liver abscesses which may occur anywhere as part of a pyæmic infection; nor with the extraneous foci, such as the "suprahepatic abscess" of Cantlie, which is said to be due to chill.

Geographical Distribution.—As a complication or sequela of bacillary dysentery liver abscess is very rare indeed, and when it does occur is probably a secondary infection from septic foci in the ulcerated gut. That the Shiga and allied bacilli are the cause of epidemic dysentery, and that it is this epidemic dysentery which occasionally visits asylums, &c., in England, will account for the rarity of liver abscess in such institutions. A like reason will explain the comparative rarity of hepatic abscess in the W. Indies as compared with India, and amongst our South African troops as compared with Egypt.

Hepatic abscess is essentially connected with endemic (or amœbic) dysentery; and therefore, as would be expected, is connected with the endemic tropical distribution of that disease.

Etiology and Pathology—**Predisposing Causes**—1. *Dysentery.*—The *Amœba coli* causing the dysentery is the active agent in the causation of the abscess; therefore, recognised or unrecognised, dysentery is the chief predisposing cause.

2. *Recent Arrival in the Tropics.*—In Waring's cases 50 per cent. occurred in people after less than four years residence.

3. *European Nationality.*—The incidence is about 35 times greater amongst Europeans than amongst natives.

4. *Male Sex.*—On an average only about 5 per cent. of those attacked are women.

5. *Middle Age.*—The condition is rarely met with below the age of 20, or above that of 50.

6. *Circumstances causing an Abnormal Condition of the Liver.*—An abscess will rarely or never develop in a normal liver, notwithstanding the fact that amœbæ may be almost constantly poured into

it from an ulcerated bowel (*Musgrave*). The following circumstances will be such as are likely to cause hepatic hyperæmia and congestion :—

(*a*) *Over-indulgence in Eating.*

(*b*) *Over-indulgence in Alcohol.*—In Waring's cases 65 per cent. were alcoholics.

(*c*) *Malaria.*—The consequent hepatic congestion and lowering of general vitality will predispose.

Specific Cause.—1. The specific cause is *Amœba coli*, either alone or in association with certain other micro-organisms.

2. The *A. coli* is constantly present in the contents or walls of the abscess, and there are no differences between these amœbæ and those found in the intestines.

3. Both amœbæ and bacteria, after some time, may not be found in the contents of abscesses in which they were present at the beginning.

4. The bacteria may produce alterations in the nature of the lesion, so that it may come to resemble a bacterial abscess.

5. The associated bacteria are—

S. pyogenes aureus, ...	frequent.
B. coli, ...	frequent.
B. pyocyaneus and others, . . .	rare.

6. Bacteria are more commonly associated with amœbæ in multiple than in solitary abscesses.

Method of Infection.—This has been a source of much discussion. It seems fairly certain that—

(*a*) In multiple abscesses the portal circulation transmits the infection.

(*b*) In solitary abscesses the same channel is probably the usual one; but, in addition, the amœbæ may sometimes make direct peregrinations through continuous tissue.

In almost every case the evidence is conclusive that the abscess is secondary to amœbic infection of the colon. There are some cases, however, in which the abscess would appear to be primary, since careful search shows no lesion of the bowel. In these cases there are three possible explanations:—

1. *Passage per Gall Ducts.*—This is untenable, for bile is toxic to amœbæ.

2. *Penetration of Bowel without Lesions.*—This is possible, in view of Schaudinn's observations.

3. *Slight Ulceration and Subsequent Repair.*—This is the most satisfactory of the explanations, as it is well recognised that even extensive lesions can be repaired sufficiently to elude macroscopic evidence.

Location and Number of Abscesses.—(*a*) In over 90 per cent. the right lobe is involved.

(*b*) In over 70 per cent. the right lobe alone is involved.

(*c*) In 50 per cent. the abscess is a solitary one.

(*d*) The most common of all conditions is a solitary abscess in the dome of the right lobe.

(*e*) In the majority of cases abscesses are rather close to the surface, whichever lobes are involved.

(*f*) The size may vary from a diameter of 1 millimetre to that of the whole lobe which remains as a shell.

Course and Termination—1. *Multiple Abscesses with Mixed Infection.*—These, as a rule, develop rapidly, do not perforate, and end in death with a septic temperature.

2. *Multiple Pure Amœbic Abscesses.*—There is a slower course, less toxæmia, and death may occur from intercurrent trouble, with or without perforation.

3. *Solitary Abscesses.*—Course still more chronic, and fair health may even be maintained for a year or more.

4. *Termination.*—(*a*) Septicæmia. (In mixed infections.)

(*b*) Encapsulation or absorption. (Rare.)

(*c*) Perforation into—

1. Right lung. } (Most
2. Right pleura. } common.)
3. Abdomen.
4. Skin.
5. Intestine.
6. Stomach.
7. Vena cava.
8. Pericardium.
9. Psoas muscle.
10. Kidney.
11. Bladder.
12. Gall bladder.
13. Spleen.

5. *Case-Mortality.*—This varies from 60 to 80 per cent., according to whether cases are operated on or not.

6. *Prognosis.*—In the case of a single abscess, recognised and properly dealt with, the prognosis is not unfavourable. In multiple, inoperable, and septic cases it is very bad.

7. *Duration.*—Varies from three weeks to a year or longer; but is generally a few months.

Symptoms.—These are extraordinarily varied. Diagnosis is consequently very difficult in many cases. Fever, enlargement of the liver, pain, and leucocytosis may all be marked without the presence of an abscess. On the other hand, a large abscess may be formed with practically no symptoms. The following are the usual manifestations:—

1. *Physical Signs.*—The liver is enlarged; usually the right lobe, and upwards (*Osler*). This sign is not invariable.

2. *Pain.*—In solitary abscesses there is usually a general soreness and tenderness on pressure. Local pains most pronounced in superficial abscesses. Radiating pains, in deep or septic conditions. In amœbic infections, pain is rarely acute, and not infrequently referred to the shoulder as in mixed infections.

3. *Jaundice.*—Rare in solitary abscess, and not frequent in multiple ones. A sub-icteric tinge is common, but is equally so in non-suppurative hepatitis.

4. *Bowels.*—The number of stools is usually increased during the abscess development.

5. *Fever.*—This is not characteristic. It sometimes remains normal throughout. The fever may be remittent, intermittent, or continuous. Septic temperatures are due to mixed infections (Fig. 49). Rigors and profuse sweats are usual in septic cases.

6. *Facies.*—The expression is frequently haggard and anxious. The features are rather drawn and pinched. The skin has a greyish-green look, and the conjunctivæ are faintly icteric.

7. *Blood Count.*—The following are the calculations of *Rogers* made from Calcutta blood examinations:—(*a*) Absolute leucocytosis is nearly always found with amœbic abscess; but in chronic cases with marked anæmia only a relative leucocytosis may be found. (*b*) The degree is variable, being highest in the most acute cases; a low degree being met with in cases with insidious onset. (*c*) In acute hepatitis without suppuration both absolute and relative leucocytosis is absent.

Musgrave, however, considers that undue importance is given to the differential count, since the intestinal infection alone may be responsible for a leucocytosis of 20,000 or more for some days or weeks.

Treatment.—This is purely surgical. The diagnosis being established, the patient should be anæsthetised without delay.

Exploration should then be made with a full-sized aspirating needle. If there are local signs, such as a tender spot, or local bulging, then that situation should be explored with the needle.

If there is no indication of the situation, then insert in the eighth or ninth interspace, in the right axillary line, about an inch from the costal margin, and well below the limit of the pleura. Insert inwards, upwards, and backwards. If no pus is found try a fresh spot. At least six punctures should be made before abandoning the search.

Having found pus, the next thing is to operate.

The following is the usual English operation:—

1. *If Pus is found below Costal Border:*—(*a*) Aspirating needle is left *in situ*.

(*b*) Three-inch incision is made in abdominal wall.

(*c*) If no adhesions are discovered between the abdominal wall and liver, the capsule of the latter must be secured to the former by a double row of stitches.

(*d*) In either case a pair of Grim's forceps is run along the needle, and pushed into the abscess.

(*e*) Open the forceps' blades a little, and then withdraw both them and the aspirating cannula.

(*f*) Find out the size of the cavity with the forefinger.

(*g*) Insert a drainage tube, and, when pus has finished flowing, apply an antiseptic dressing and bandage.

2. *If Pus is struck through an Intercostal Space:*—(*a*) If more room is wanted, resect some rib, and stitch the diaphragm to the thoracic wall and skin.

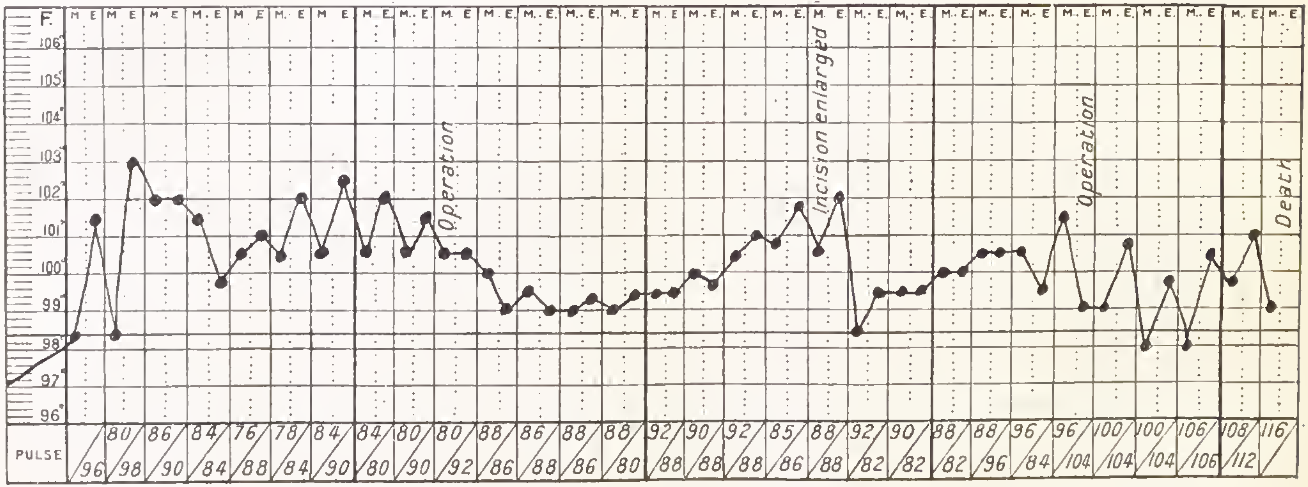


Fig. 49.—Liver abscess—European male, aged 31 years—Death. (One deep abscess of right lobe was found and drained at the first operation. At the *post-mortem* examination a second abscess was found deep in the left lobe.)

(*b*) If the pleura is accidentally opened, take no notice of the pneumo-thorax, but stitch the opening before dealing with the abscess.

There are several alternative methods of which the following has often good results:—

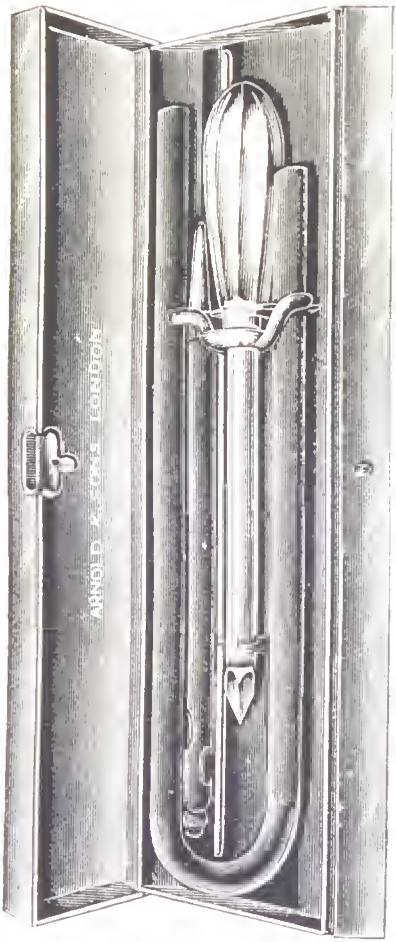


Fig. 50. Liver abscess trocar (Manson's), latest pattern, with metal end drainage tube, introducer, and spare India-rubber tubing, in metal nickel plated case.

Manson's Operation.—The *modus operandi* of this, is to find the abscess with an exploring needle as before.

Then make a 1-inch skin incision. Plunge in a ⅜-inch trocar (Fig. 50) and cannula. Remove the trocar.

A drainage tube (previously stretched on a stilette by pressure against two terminal buttons) is then pushed through the cannula to

the back of the abscess. The cannula is withdrawn, and the near button slipped off the free end of the drainage tube; still holding the distal end at the back of the abscess by means of the stilette. The result of this manœuvre is that the elastic tube contracts, and completely fills the passage into the abscess.

The stilette can now be withdrawn, and the free end of the drainage tube cut to a suitable length, transfixed with a safety-pin, and the latter fixed to the skin with plaster.

Treatment after Operation.—While the discharge is free, the dressings should be changed twice daily.

In satisfactory cases it will generally be found that the discharge lessens considerably in a day or two, in which case the dressings need be changed less often. If no further abscesses are present, and the drainage is good, the temperature generally falls to normal.

A rise of temperature, due to improper drainage, may necessitate a further operation to enlarge the sinus, or make a counter-opening.

A rise of temperature, due to septic infection, will indicate that the cavity should be washed out daily with antiseptics.

A rise of temperature, other than from these causes, may point to the existence of other abscesses. These should be at once sought for with an aspirating needle, and, if found, opened and drained.

Abscesses discharging through the lungs should be operated on to secure external drainage if:—

1. The temperature remains high.
2. The pus output does not diminish.
3. There is loss of body weight.

Otherwise it is probably better to leave them alone.

General Remarks.—Great care should be taken to prevent the access of septic matter.

The diet should be light, but nutritious.

The bowels should be kept open.

For restlessness, sleeplessness, or pain, opium is indicated.

A quinine mixture is often of use.

Great quiet, care, and warmth are essential until convalescence is well advanced.

After recovery, residence in a temperate climate is advisable; but patients obliged by duty to remain in, or return to, the tropics, have often done well for many years after; but, in such cases, the utmost care and circumspection is necessary.

CHAPTER XXVI.

KALA-AZAR.

(*Black fever.*)

Definition.—A chronic infective disease of warm climates, characterised chiefly by—(1) Splenic hypertrophy; (2) irregular pyrexia uninfluenced by quinine; (3) certain gastro-intestinal symptoms, such as diarrhœa and dysentery.

Synonyms.—*Tropical febrile splenomegaly; Dum-dum fever; Burdwan fever.*

History and Geographical Distribution.—The first notice we find taken of the disease known as kala-azar (and now recognised as a specific entity) is in 1875, when an epidemic began near the Paro Hills in Lower Assam. For the next twenty-five years it continued to appear (chiefly during the hot rainy season of April to August) in that district, keeping chiefly to the south bank of the Brahmapootra, depopulating whole districts, and causing the death of about one-fifth of the population.

An exceptional exacerbation of this Assam epidemic occurred in 1887, and extended over a large area of the country. The tendency of the disease seemed to be, that it followed lines of human intercourse, and was only stopped by the intervention of uninhabited jungles. As the epidemic swept on, a less infective, but permanent, endemic focus remained behind.

Many theories as to its nature have been propounded—in nearly all cases it was considered as a form of malaria, or malarial cachexia. In addition to this theory one Indian Government Commissioner (Giles) regarded it as malaria plus ankylostomiasis; another observer as a virulent type of malaria with a secondary infection. Bentley thought it a grave form of *M. melitensis.*

Finally, in November, 1900, *Leishman*, while examining some spleen smears from a military patient who had been invalided from India with Dum-dum fever, and had died at Netley, came across some minute spherical or oval bodies. These observations he published in the *British Medical Journal* of May, 1903, suggesting that they were possibly an immature form of trypanosome—possibly allied to *T. gambiense*, then recently discovered.

In the winter of 1902-3 Leishman observed in smears from the internal organs of a trypanosoma-infected rat some bodies much resembling those he had found in the case quoted above, and, moreover, these spherical or oval bodies possessed two chromatin masses as do the trypanosomes.

Shortly after this *Donovan* found the same parasites in Indians, by splenic puncture. They were also found by *Marchand*, of Leipsic, in spleen sections from a German soldier who had died of a spleno-megaly fever, apparently acquired at Pekin.

Since then they have been reported from the Soudan and Algiers; *Rogers*, *Bentley*, and *James* have demonstrated them in kala-azar patients from Assam; *Airde* has found them in a patient in Hankow (China); *Freer* in two Indian labourers at Penang; *Manson* in a European patient invalided home from India; and *Musgrave* in Manila. Thus, while not, up to the present, much investigated, the disease has undoubtedly a wide tropical and sub-tropical range.

An interesting feature of these bodies is the re-discovery of them by the American observer, *Wright*, in the chronic ulceration known as Granuloma endemica (Oriental sore)—see Chapter xxiii.

Etiology and Epidemiology.—Kala-azar attacks any race, sex, or age, and, unlike the affection of malaria for the new-comer, it has a predilection for the acclimatised native or old resident.

Although somewhat chronic in type, it is exceptionally fatal; the case-mortality being usually about 96 per cent. (*Rogers*), either due to exhaustion or to intercurrent disease, especially dysentery.

As the method of propagation is at present unknown, it is highly important that all known details should be recorded in order that investigators may gather the lines on which to proceed.

1. The disease proceeds often in epidemic form, clinging to a place for some years, and leaving behind a modified endemic heritage.

2. The infection of a new area can usually be traced to an imported case.

3. The inmates of the house in which he lodged are usually the first to be attacked.

4. The house appears to retain the infection for many months.

5. The parasite *Leishmania donovani* (Leishman - Donovan bodies, Fig. 51) is present in the spleen of every case of kala-azar; as also in every case of granuloma endemica.

6. The distribution of these two diseases is different.

7. The parasite has only rarely been observed in the peripheral blood (two moribund cases), and they were then found by *Stephens* and *Christophers* to be of typical structure, but all included in leucocytes.

8. The fever in kala-azar bears no relation to the number of parasites found.

9. *Rogers* has shown—confirmed by *Leishman* and *Christophers*—that if infected blood or spleen pulp be mixed with a 5 per cent. citrate of soda solution, acidified with normal citric acid, and kept at a low temperature (22° C.) that the parasites will multiply, increase in size, and ultimately develop into long, slender, motile, flagellated organisms, differing only from the known trypanosomes in that they have no undulating membrane, and that the flagellum springs directly from the nucleated end of the body.

The following are the chief *features of the parasite*:—(*a*) They are circular or oval; 2·5 to 3·5 μ in size.

(*b*) They are clearly outlined, and apparently possess a cuticle, as they are rarely distorted in films.

(*c*) Two chromatin masses are found—a larger one which forms part of the periphery of the parasite, and stains lightly with Romanowsky; a smaller one, usually opposite the other in the short axis of the parasite, which stains intensely with that stain.

(*d*) Most parasites contain one or two vacuoles displacing the cytoplasm towards the periphery.

(*e*) In smears from the spleen or splenic punctures some of the parasites are found free, but the majority are in leucocytes or in fragments of splenic cells (resembling unaltered red cells).

(*f*) The majority of the parasites are found in the large hyaline or finely granular cells (macrophages); a smaller number in endothelial cells; less still in large mono-nuclears; and least of all in polymorpho-nuclear leucocytes.

(*g*) Development takes place by division at the thick end. The large masses divide first; and much later, the small chromatin mass. Three to six bodies are thus formed, the large nuclei being at the periphery.

The *problems* now connected with this disease are, therefore:—How does the parasite leave the body; what is the intermediate host; how is it acquired; and what is the connection with granuloma endemica?

The fact that a low temperature is favourable to propagation would seem to indicate that some cold medium, such as soil, water, or a cold-blooded animal, is the first step in its extra corporeal life. The infectivity of a newly-infected house would rather point to some such intermediate host as *Cimex lectularius*, which inhabits the house itself, than to such personal parasites as fleas or migratory pests as mosquitoes.

That the parasite has been but rarely found in the peripheral blood is no argument against this hypothesis, for the yellow-fever germ is conveyed by *Stegomyia* from a patient's blood, yet has, so far, eluded microscopical investigation. A possible analogy is that of malignant malaria, in which the early forms of the parasite are alone found in the peripheral blood: while the sporulating—the later developmental forms—are only found in the spleen and other internal organs. Even thus, there may be a yet unrecognised early form of the parasite circulating in the peripheral blood in cases of kala-azar.

With regard to the possible connection between kala-azar and granuloma endemica (*Manson*), in view of the fact that the former disease is malignant and the latter benign, hazards the suggestion that the reduction in virulence in the latter disease might possibly be effected by passage through the camel (which is said to have a like distribution) in the same way that the virus of smallpox is

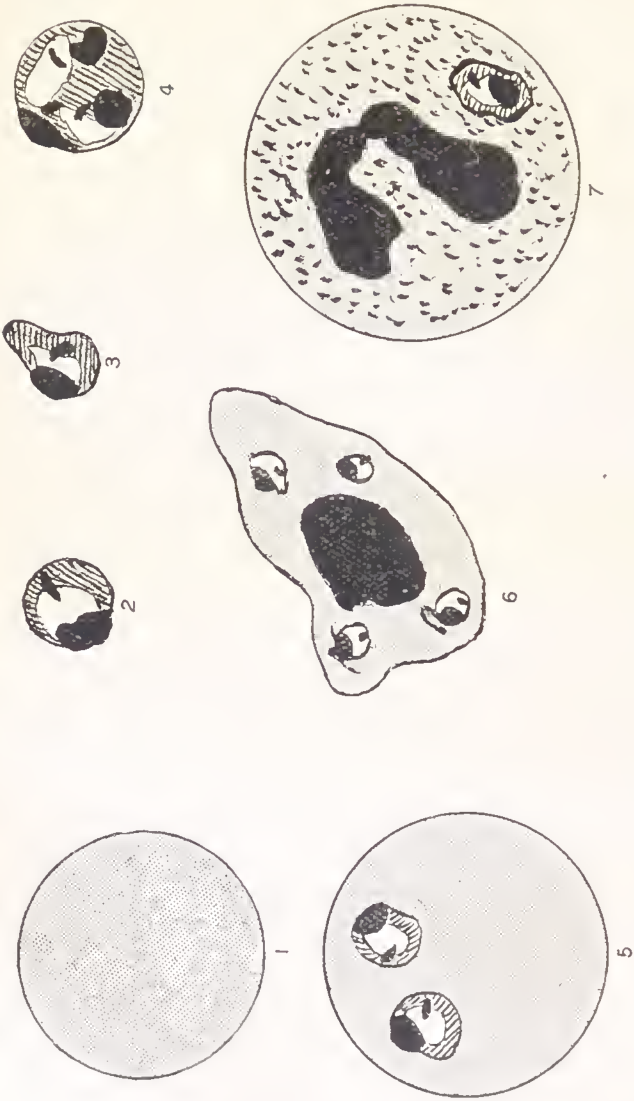
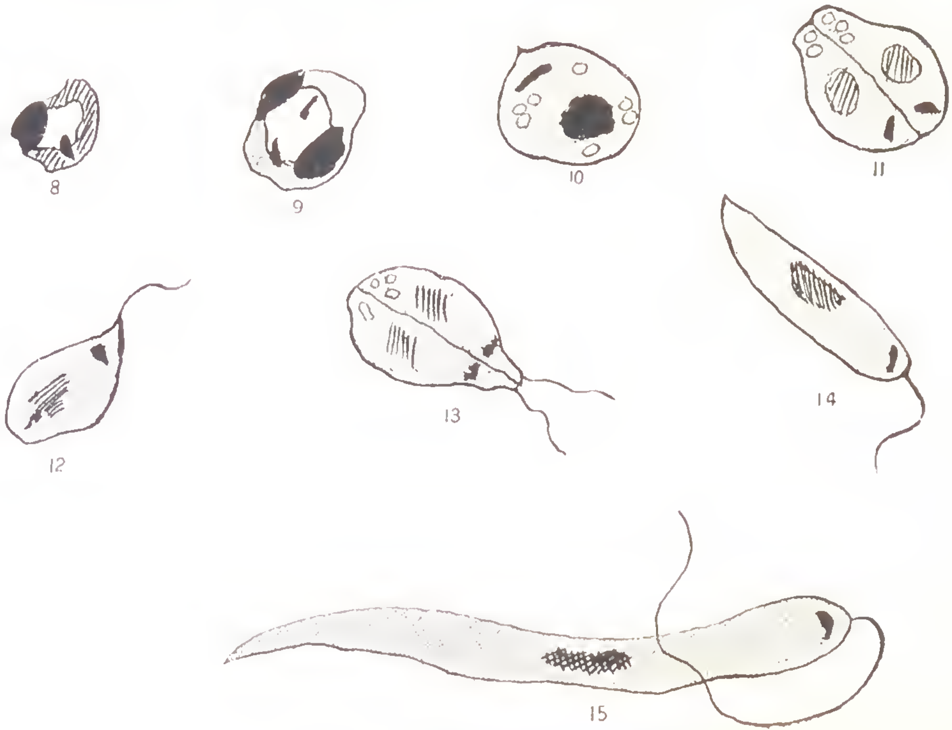


Fig. 51.—Leishman-Donovan bodies. (*Leishmani Donovani.*)

1. Normal red corpuscle.
2 and 3. Types of parasite (showing two nuclei and vacuole).
4. Dividing form.
5. Two parasites in apparently unaltered red cell.
6. Parasites in large mono-nuclear leucocyte.
7. Parasite in polymorpho-nuclear leucocyte.

8-15. Development (after *Leishman*) of the parasite, in sodium citrate solution, showing the development of trypanosome form, but differing from trypanosomes by having a purely terminal flagellum at the micro-nuclear end, and in having no undulating membrane



deprived of its virulence by passage through the cow, and that the virus of granuloma endemica might protect against kala-azar.

Symptoms.—The chief **clinical characters of the fever** are:—

1. *Pyrexia.*—This is of an irregular and continued character, lasting for several weeks, and recurring frequently with short intervals of apyrexia.

2. *Enlargement of Spleen.*—This is frequently very great, and may extend below the umbilicus. It is the most distinctive character of the disease (*Stephens* and *Christophers*).

3. *Emaciation and anæmia* occur after the disease is thoroughly established.

4. *Abdominal Symptoms.*—Dysenteric ulceration is common, especially in the later stages.

5. *Leucopenia.*—The leucocytic count generally falls to about 2,000 per mm. In the cases of Stephens and Christophers the relative percentages of the leucocytes have not been found to vary much from the normal, though Freer, in two cases at Penang, found the percentage of polymorpho-nuclears very much below the average, while that of the lymphocytes and large mono-nuclears was relatively increased—possibly merely a coincidence.

6. *Phagedænic Processes.*—Cancrum oris, noma vulvæ, &c., are common. Ulcers may occur about the knees, elbows, or legs. All the phagedænic cases investigated in Madras up to the present have yielded parasites on splenic puncture.

7. *Skin eruptions* of a papular nature frequently occur, in advanced cases, about the thighs and scrotum.

8. *Hæmorrhagic symptoms*, such as purpura, petechiæ, or epistaxis, may be found.

9. *Œdema*, especially of the feet, may be occasionally present.

10. *Pigmentation of the skin* (to which the term "Kala-azar" is due) has been reported, but does not appear to be common.

The usual **clinical picture** of a case is as follows:—After a minimum of prodromal symptoms, a rigor, or, more rarely, emesis, may usher in a febrile attack, often severe. The attack lasts for several weeks, and is accompanied by enlargement of spleen and liver. The type of fever is remittent; it is generally followed by a short spell of apyrexia and general improvement. More fever and splenomegaly follow, and thus febrile and non-febrile periods follow each other, unchecked by quinine, until a pyrexial cachexia is established, the temperature being continuous, though not very high.

Emaciation and anæmia contribute to the earthen pallor of the skin. Petechiæ, epistaxis, or gangrenous conditions may make their appearance as the disease advances, and patches of œdema are not uncommon. The cachectic condition with progressive enlargement of the spleen, emaciation, and anæmia, may continue for many months until asthenia or intercurrent disease carries off the patient. Diarrhœa and dysentery are especially common complications.

The prognosis is very gloomy, as the case-mortality is about 96 per cent.

Diagnosis.—Some difficulty may arise in excluding *Malta fever*, owing to the similarity of the chronic and irregular type. In

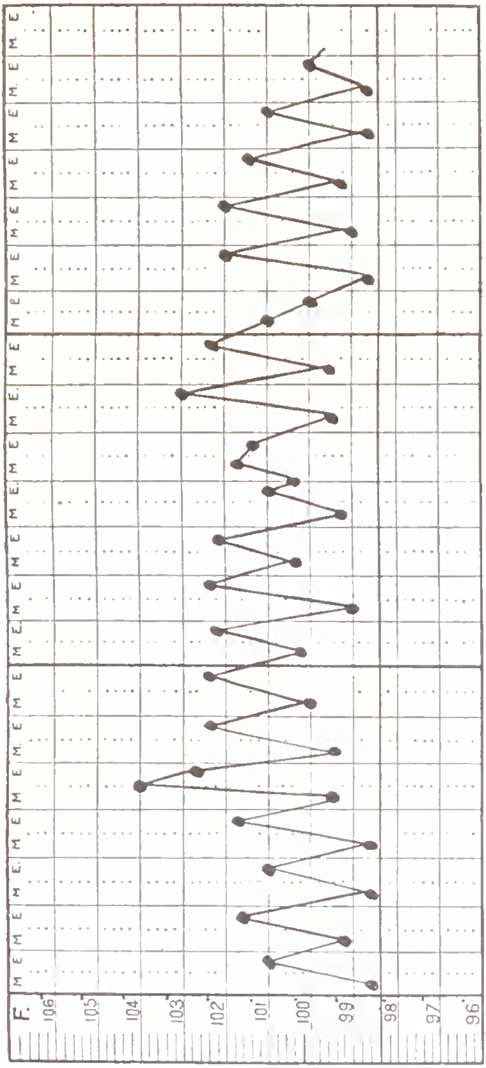


Fig. 52.—Temperature chart in an advanced case of *kala-azar*. Spleen reaching to umbilicus. (After *Stephens and Christophers*.)

kala-azar, however, there is much more enlargement of the spleen; there is absence of the joint rheumatism so characteristic of Malta fever; and there is not the same profuse sweating that occurs in the

latter disease. Both are unamenable to quinine, and both are associated with profound anæmia. In kala-azar, however, the emaciation is more progressive and obvious, and the fever eventually becomes continuous, instead of gradually ceasing (Fig. 52) as in Malta fever; but the seriousness of the diseases is very different—a case-mortality of about 2 per cent., as compared with 96 per cent.

The locality from which the patient comes should be taken into account; and it might even be necessary to resort to splenic puncture to clear up the diagnosis.

Malaria can be excluded by the type of fever, the uselessness of quinine, and the absence of parasites from the blood.

Leucocythæmia must be excluded. This is most important, as otherwise a splenic puncture might lead to fatal abdominal hæmorrhage.

Technique of Splenic Puncture.—1. Use a hypodermic needle.

2. Boil it first in normal saline solution.

3. Cleanse the skin.

4. Puncture between the ribs if the enlargement is not great; otherwise where it is most prominent.

5. The parasite is very little in the blood—mostly in the cells; therefore try not to take up too much blood.

6. Eject on to glass slides. Dry and stain with the Leishman stain.

Pathology and Morbid Anatomy—(1) **Macroscopic.**—The *spleen*, as if frozen, retains its shape after removal from the body. Although very firm, it is friable, and has no fibroid toughness.

The *liver* is firm, but friable.

The *large intestine* usually shows extensive multiple ulceration.

Peritonitis, *broncho-pneumonia*, and *septic infarcts* are not uncommon.

(2) **Microscopic.**—

Spleen. *Liver.* *Bone-marrow.*	Pulp-smears show immense numbers of the parasites.
Lung. *Kidney.* *Testis.* *Lymph glands.* *Intestinal ulcers.*	Smears may show the parasite, but to a less extent than the above.

If sections are cut, the following appearances will be observed:—

Liver.—Close to the wall of the lobular capillaries, within the lumen, will be seen many large, extended cells, crowded with parasites, which appear to lie in vacuoles, but are apparently part of the parasite body. The nature of the cells is doubtful, but they somewhat resemble the macrophages found in malarial organs.

Spleen.—The red cells have no contained parasites, nor are they found as free forms.

The greatest infection is in macrophages similar to those found in the liver; and a certain number of large mono-nuclear leucocytes are also found to contain parasites.

Types of Cells in which Parasites occur:—(*a*) *Modified Endothelial Cells.*—These are identical with those found in the capillaries of the testis and of granulation tissue.

They have an extensive vacuolated protoplasm with an oval nucleus.

Half a dozen or a dozen parasites may be found in them.

(*b*) *Large Round Macro-nuclear Cells.*—Found as the above, but attached to the capillary wall. They are also found in the post-mortem blood from the large veins.

They have a ground-glass, vacuolated protoplasm.

A score or more of parasites may be found in them.

(*c*) *Large Bi-nucleated Cells.*—Found in large numbers along the capillary walls in liver and spleen; in the latter their processes extend among the smaller pulp cells.

Numerous parasites may be found in them.

(*d*) *Large Asymmetrico-nuclear Cells.*—They occasionally show signs of necrosis, and stain more intensely than (*c*). The nucleus is laterally displaced, and the centre of the cell has a vacuole, around which a hundred or more parasites may be grouped.

Unless carefully fixed with osmic acid vapour, they will seldom be found unruptured in an ordinary film.

(*e*) *Bone-marrow.*—The parasites will be found in large numbers in macrophages; to a less extent in large mono-nuclears; and a few in myelocytes or polymorpho-nuclears.

(*f*) *Large Intestine.*—Parasites are found in similar cells to other situations. They occur in the early stages of ulceration, and in granulation tissue.

(*g*) *Granulation Tissue.*—Parasites will be found in the endothelial cells of the fine capillaries, in sections of papules or skin ulcers. In the small vessels cells (crowded with parasites) will be seen, similar to those in liver or spleen.

(*h*) *Lymphatic Glands.*—Parasites may be seen in the large lymph-sinus cells, and in cells of the reticulum, in glands draining the area of an infected skin-lesion.

Treatment.—In the present state of our knowledge this is, alas, highly unsatisfactory.

Drugs, such as quinine and arsenic, are of no use at all.

All we can do for a case is to advise change to a cool climate, combined with rest, and good food and hygienic conditions.

For prophylactic measures it would be well to guard against the bites of insects when in an endemic area, or when near a patient.

In the endemic region itself, it would be safer to isolate all patients, and to burn their fomites.

CHAPTER XXVII.

LEPROSY (Λεπρος = scaly).

Definition.—A chronic bacillary disease of slow incubation, characterised by the formation of neoplasms, giving rise either to thickenings and nodosities or perversions of sensation, and to progressive ulcerative mutilations.

Synonyms.—*Elephantiasis Græcorum*, *Lepra*, *Satyriasis*, *La lèpre* (French), *Aussatz* (German), *Lebbra* (Italian).

History and Geographical Distribution.—The recognition of the disease is of very ancient origin. As far as we know, Egypt was the earliest home of leprosy. A papyrus of B.C. 4260 mentions it, as do other Egyptian authors, such as *the Ebers papyrus* (B.C. 1300).

The biblical accounts of leprosy probably largely refer to other cutaneous condition, such as vitiligo, &c., although doubtless the disease must have existed in Palestine at the time.

An Indian classic of about B.C. 1400 mentions the condition.

It was recorded in Japan about B.C. 1250.

Aristotle mentions it in Europe B.C. 345 by the name of "Satyria."

Galen and *Celsus* speak of it as "Elephantiasis," and *Aretæus* as "Leontiasis."

The conquests of the Romans, and the later peregrinations of the pilgrims, were probably accountable for the diffusion of the disease throughout Europe.

Legislation against leprosy was enacted in Lombardy in the seventh century; by Charlemagne, in France, in 789; by Noel Dha, in Wales, in 950.

Leper asylums were established throughout Europe in large numbers. The first in England was founded at Canterbury in 1096, followed by 111 others in various parts of the country, of which the last was at Highgate in 1472.

The disease had practically disappeared from England before the time of Henry VIII., but in Scotland it existed till much later. The last indigenous leper was seen in the Shetland Islands in 1798, and a case occurred in Edinburgh in 1809 (*Abraham*).

In temperate Europe there are now but few endemic centres, the most important being the Scandinavian Peninsula and Iceland.

It is widely spread amongst the tropical and subtropical parts of Asia, throughout almost all Africa, the tropical belt of North, Central, and South America, and the West Indian Islands.

Etiology.—Leprosy is due to a specific bacillus, the *B. lepræ*, discovered by *Hansen* in 1871, and more minutely studied by *Neisser* in 1879.

The following is the morphology, &c., of this bacillus:—

1. Delicate slender rods, 5 to 6 μ long (tubercle 2 to 4).
2. Protoplasm aggregated into beads.
3. Stains with any simple stain, but Ziehl-Neelsen usually employed.
4. Stained by Gram.
5. Non-motile.
6. Does not liquefy gelatin.
7. Spore formation not known.
8. Very hard to cultivate.

Emile-Weil, of the Pasteur Institute, has been successful with the following medium:—

Glycerin,	40 grms.
Glucose,	8 ,,
Peptone,	10 ,,
Agar,	20 ,,
Yolk of egg,	19 ,,
Broth, to	1 litre.

Growth commences on the fifth day and continues for fifteen or twenty days, when it stops, and cannot be renewed by subculture.

9. The bacilli are found in all leprous tissues and discharges.
10. They exist both free and in the cells.
11. They are found in lumps and clusters (not singly like the tubercle bacillus).

That the disease has spread from country to country, and then gradually tended to die out, is a matter of history and observation; but the channel or channels of infection are even still shrouded in mystery. From remote times it has been considered to be contagious, but the weight of evidence has been far from proving this as the only, or indeed the chief, means of communication.

Neither race, temperature, humidity, nor altitude have apparently anything to do with the incidence of the disease.

Poverty, as in the case of most other endemic diseases, seems, as would be expected, to have a predisposing effect.

Vaccination has been brought forward as a means of spreading the disease, but the few alleged cases of transmission by this means are open to serious doubt, and "no danger need be apprehended from the vaccine lymph even of a leper, provided he be vaccinated on healthy skin" (*Acland*).

The *male sex* shows a greater predisposition to the disease than the female. With regard to *age*, the disease develops most frequently between the ages of 15 and 30. It may appear, however, at extremes of life.

Galloway, in Singapore, quotes a case in his practice of the appearance of leprosy in a Chinese boy, aged 2½ (1887). There

was no other disease in the family, but the Ayah was found to be leprous. Five and a-half years later the disease was quite advanced.

Abraham quotes cases seen in Norway in which patients exhibited signs of the disease at about the age of 80.

It remains, therefore, with regard to leprosy, to consider in detail the five methods by which any disease is usually communicated from one person to another :—

1. *Transmission by contagion* (*i.e.*, the inoculation of some loss of epidermal continuity or accessible mucous membrane by contact with a diseased person or by contact with effects soiled with emanations from such person). It has been maintained that the contagiousness of leprosy is proved by the diminution of the disease after the segregation of lepers during the Middle Ages, but this statement goes for nothing, as the isolation was never absolute, nor were antiseptic precautions known. Moreover, if the disease is endemic in any country or district, it is extremely hard to say that any given case has been communicated by contagion, since there may be so many other ways by which the virus may have been conveyed. Out of eight cases of the kind brought before the Leprosy Commission in India only one would bear examination—viz., a sweeper in the Calicut Asylum who contracted the disease after attending inmates for twenty years. Other cases brought forward to support the contagion theory are :—A man in Trinidad, who developed the disease two years after living with a leprous woman ; a cook in the leper's home at Jamaica, who frequently slept there, and exhibited the disease after seventeen years.

Cases of the same nature have been reported from India, South Africa, Cyprus, Russia, Brazil, &c. Then we have the cases of Fathers Damien and Gregory in Molokai, and two French Sisters in French Guiana and Tahiti, who are said to have contracted it by needle-pricks while sewing lepers' clothes.

In the whole of this evidence there is nothing to prove that the disease was not conveyed by food, or by insect-bites, or by air. In fact, there is a preponderance of negative evidence, since the attendants in hundreds of asylums have never contracted the disease; cohabitation with lepers has often been indulged in without evil results ; post-mortem wounds have not resulted in an infection.

Even the most conclusive cases of the contagion hypothesis will bear no weight—*e.g.*, *Benson's* case of an Irish soldier who returned from India with leprosy ; his brother, who had never left Ireland, slept with him for a year and a half, and three years later showed signs of the disease, and, later, died of it.

Wolff, of Strassburg (1904), recorded a case of leprosy contracted in Tonkin, who lived with a nephew for two months, the latter eventually exhibiting signs of the disease. In neither of these cases is the contagion theory conclusive, for the disease might have been communicated by food, by the air, or by an intermediate host.

Some writers seem to consider that because the disease is evidently distributed by human intercourse, that it must perforce be

contagious. This is, of course, illogical. Other similar cases have been brought forward, such as *Lorand's* in Sweden and *Thorp's* in Norway, and *Hellat's* in Russia, all of which are equally open to the same objection.

If it is contagious at all, it must be in a very slight degree—far less so than tuberculosis (*Abraham*).

2. *Infection by the Air.*—Bacilli are extruded in enormous numbers from the nasal secretion of lepers, and must either remain in the air or reach the soil. *Sticher* is of the opinion that in leprosy the primary effect has always its seat in the mucous membrane of the nose, and that leprosy, therefore, is a primary nasal disease, in an even more limited sense than syphilis is primarily a sexual disease, and tuberculosis an apical lung disease. The lymphatic glands of the nasal mucous membrane are connected with those of the skin, with those of the sub-arachnoid spaces of the brain, and with those of the spinal column and the peri-neural sheaths of the peripheral nerves, by which channels the bacilli could invade the whole body.

This hypothesis is highly improbable, however, since, were the disease air-borne, there would be a considerable incidence in the neighbourhood of those asylums situated in non-endemic countries, as in the case of smallpox. *Hillis*, indeed, reports 60 cases occurring in healthy persons in the vicinity of Mahaica Asylum, but as that place is an endemic centre the argument is worthless.

That cases may be transmitted directly from a patient to a healthy individual, by transference of infected nasal mucus by sneezing or otherwise, is quite a likely event, and may account for a certain number of infections

3. *Infection by Heredity.*—A fairly widespread idea that the disease is hereditary has been, and is still, held both by medical and lay people. Doubtless the reason for this belief is founded on the occasional occurrence of cases of leprosy amongst members of one family.

The *Indian Commission*, in investigating this matter, could only find family histories in about 5 or 6 per cent. of the cases under review.

Wright, in his work on *Leprosy an Imperial Danger*, quotes six cases occurring in a Prince Edward Island family; but the same account gives also five other infections amongst those immediately connected with the family by marriage or otherwise.

Bibb reports two cases from Mexico in which infants were removed, after birth, from leprous mothers to healthy surroundings in leprosy-free districts, and yet developed the disease at or after 20 years of age.

At Molokai, amongst 2,864 incarcerated lepers, not more than 26 children there born were found living after a period of 18 years, and of these only two were lepers.

There is no authentic instance of a child ever having been born a leper.

From the consideration of all these points it is, therefore, highly improbable that the disease can be ascribed to heredity; and even in those cases which seem most favourable to the theory, it is impossible to exclude a possible infection by air, food, or intermediate hosts.

The evidence against it is very strong, in support of which may be quoted *Hansen's* investigation of the 160 Norwegian lepers who had emigrated to the United States, and amongst the descendants of whom, even to their great-grandchildren, he could find no trace of leprosy.

It is, however, highly probable, as in the case of tuberculosis, that an inherited disposition may exist in certain families.

4. *Infection by Diet.*—To vegetable, pork, fish, and other diets leprosy has been frequently attributed by various writers, such as *Munro*, *Hutchinson*, and others. It has also been ascribed to the use of contaminated water by *Liveing* and others.

Of these the fish theory, so ably championed by *Hutchinson*, has probably met with most support. His theory suggests that either the bacillus itself may infect decomposed fish, or that the fish-substance may excite the tubercle bacillus to unusual forms of development. To support it he points out—

(*a*) That coast-dwellers are most affected;

(*b*) That it is more prevalent amongst Roman Catholic communities, who have a prescribed fish diet;

(*c*) That vegetarian Brahmins are almost exempt.

He claims that this theory explains the antiquity of leprosy, its widespread distribution, its prevalence in Roman Catholic countries, the disappearance of the disease with the advance of civilisation, and its absence in Central Russia. Hutchinson's investigations in India and South Africa resulted in obtaining a few facts which apparently supported the idea, but most investigations, while not perhaps excluding the possibility of this channel of infection, certainly exclude the probability.

In the first place, the bacillus has not been demonstrated in fish. In the second place, it is rare for natives anywhere to eat uncooked fish, and cooking should certainly kill the bacillus. Thirdly, fish not eaten fresh is usually salted, and sodium chloride has been shown by *Rost* to be inimical to the life of the *Bacillus lepræ*. Fourthly, why, in places such as the Orkneys and Shetlands—formerly an endemic focus of leprosy—does the disease not still exist, since the inhabitants still habitually consume large quantities of decomposing fish, as they did in the days of leprosy? Further, there are many places where leprosy is present and where fish is never eaten (*Hansen*). Lastly, how is it that amongst the millions of fish-eating natives, most of whom eat more fish than any Roman Catholics, there is such an extremely small relative incidence, while it is very rife amongst many Roman Catholic communities (*Hutchinson*)?

We can conclude, therefore, that if occasionally fish eating may

be an etiological factor in the propagation of leprosy, it must indeed be a rare one.

5. *Infection by Intermediate Hosts.*—This method is, strictly speaking, merely the process of contagion carried a step further; that is to say, instead of the inoculation taking place by contact it is secured by the agency of some insect or parasitic intermediary.

It remains to consider whether this channel is possible or probable in the case of leprosy.

We may, for practical purposes, neglect pests other than the following:—Ticks, flies, lice, mosquitoes, fleas, and bugs.

Ticks.—The distribution of the biting ticks, as personal acquaintances of man, is too limited to be thought of for a moment as a causal factor, other than in the rarest of instances.

Flies.—The distribution of the biting flies must, generally speaking, put them out of court. The common house fly and other allied species of *Musca* could, of course, only act as mechanical carriers of the disease. They would first have to gain access (an easy matter) to infected sources, such as leprous nasal mucus or cutaneous ulcerations. They would then (not such an easy matter) have, while still contaminated, to infect the mucous membrane or skin abrasion of some healthy person. That this method could be at all probable seems very unlikely; moreover, the flies examined by the Indian Leprosy Commission gave negative results.

It is just possible that the contaminated flies might infect foodstuffs; but against this we have the fact that the bacillus has not been isolated from foods, and, further, that the disease does not manifest itself as a primary lesion of the mouth or alimentary tract.

Lice.—*Daniels* has suggested lice as possible hosts in the diffusion of beri-beri.

With leprosy, although perhaps possible in isolated instances, it may certainly be considered as a negligible factor.

Mosquitoes.—These and allied insects have long been looked upon, in Japan and elsewhere, as possible sources of infection in leprosy.

The *Indian Leprosy Commission* examined many mosquitoes with negative results.

Arning, in the Sandwich Islands, examined mosquitoes full of blood sucked from severe cases of cutaneous leprosy, but was never able to find the bacillus either in or on them.

Jeanselme's investigations, in Indo-China, were also negative.

In 1906, however, *Goodhue*, of Molokai Leper Settlement, at last demonstrated the *Bacillus lepræ* in the mosquito (*Culex pungens*). Mosquitoes, therefore, now come within the pale of practical politics.

From what we know, however, of these insects as carriers of disease, only a limited number of definite species are capable of acting as hosts to one type of parasite—*e.g.*, certain of the genus Anopheles will alone convey the animal parasite of malaria; the embryos of the nematode *Filaria nocturna* have only been proved to be carried by certain few species of three genera—*Colex*, *Panop-*

lites, and *Anopheles*. Of the presumably bacterial diseases yellow fever is only carried by *Stegomyia fasciata*, dengue probably only by *Stegomyia fasciata* and *scutellaris*.

In such cases, therefore, the incidence of the various diseases will be determined by the distribution of the intermediate hosts. Now, leprosy does occur, or has occurred, over a very large portion of the earth's surface—frigid, temperate, and tropic; and since this area is of far greater extent than the distribution of any known genus of mosquito, it is reasonable to suppose that that insect can play no habitual part in the dissemination of the disease. Moreover, were mosquitoes to blame the incidence among physicians, nurses, and attendants on the sick would be unusually considerable, instead of being practically unknown as at present.

Fleas.—The *Bacillus lepræ* has not yet been isolated from fleas. Knowing as we do the part played by fleas in the dissemination of rat-plague amongst human beings, it is of great interest to note here that *Dean* in 1903, and, independently, *Stephansky* at Odessa, discovered certain rats to be affected with a leprosy-like disease, with neoplasms in the skin glands, &c. The bacilli isolated were indistinguishable from those of leprosy in microscopical character, regional distribution, difficulty of culture, and acid-fast staining reactions.

Rubinowitsch has found the same in Berlin rats (1903), and *Tidswell* in New South Wales.

Further investigation will, therefore, be awaited with interest.

Bugs.—After reviewing all the usual methods of transference of disease, it would seem improbable that any of them play any considerable part in the dissemination of leprosy. But the case of the bed-bug (*Cimex lectularius*) seems to have more in its favour than any of the other etiological theories:—

(*a*) *Goodhue*, of Molokai, has found the *Bacillus lepræ* in *Cimex*.

(*b*) The distribution of *Cimex* is practically universal.

(*c*) Since the sixteenth century hygienic principles and sanitary righteousness have been more and more practised in Europe. Personal and domestic cleanliness have been more indulged in, doubtless to the disgust of the bed-bug. This fact, together with the attempt at segregation of lepers, would tend to have diminished the infected material for the bugs to feed on, as well as diminishing the number of bugs who might be the potential agents in diffusion. Hence the gradual disappearance of leprosy from Europe can be explained.

Taking this as a possible explanation, many otherwise inexplicable details are cleared up. For instance, the immunity of officers and nurses in asylums. The bug frequents his room or bed, and only comes out to feed at night. The officers and nurses do not sleep with or in the same room as the patients, and, consequently, are not liable to be bitten by infected bugs.

Smyth, of Lebombo, referring to leprosy, which is endemic in his diocese, points out that the cases are sporadic in each village. The one or two lepers live in a hut apart from the rest. During the day

they eat and drink out of the same pots, and freely intermingle with the other villagers. If the leper is married sexual connection is indulged in during the daytime, but at night no one sleeps in the same hut as the lepers. Cases of leprosy which occur can nearly always be traced to mining or other labour, where the workmen are huddled together in common sleeping places.

Again, the cases which occur amongst Europeans, either when in India, or after leaving that country, are nearly always amongst those men who have lived with native women.

Hutchinson remarks that it seems improbable that the prick of an insect should convey a disease respecting which experimental inoculation always fails.

It is certain that *Dianelssen*, *Bargilli*, and others have tried inoculation experiments without success. *Arning's* experiment with the condemned Hawaiian criminal Keanu, in 1885, is unfortunately not conclusive, since other members of his family proved to have been leprous. It is, however, practically certain that the disease which resulted was due to the inoculation, for within a month painful swellings occurred along the median and ulnar nerves of the inoculated arm, and within six months they had developed into unmistakable leprous tubercles, while he died of leprosy six years after inoculation.

The following is of interest:—

REPORT OF LEPROSY COMMISSION IN INDIA, 1890.

CONCLUSIONS.

1. That leprosy is a disease *Sui generis;* and not a form of syphilis or tuberculosis, although having striking etiological analogies with the latter.

2. That it is not diffused by hereditary transmission, and that, owing to induced sterility among lepers, the disease has a tendency to die out.

3. In a scientific classification leprosy must be regarded as contagious and inoculable, but propagation by these means is exceedingly small.

4. It is not directly originated by any particular food, by climatic or telluric conditions, nor by insanitary surroundings; nor does it peculiarly affect any race or caste.

5. Poverty, bad food, and insanitary surroundings predispose to the disease, and thus have an indirect influence.

6. The method of origination is unknown.

Symptoms.—The clinical manifestation of leprosy is in two distinct forms, thought by some to be due to varied virulence of the bacillus:—

I. *Lepra tuberosa* or nodular leprosy (Fig. 53).
II. *Lepra maculo-anæsthetica.*

Incubation is very indefinite. The Hawaiian criminal who was inoculated, showed definite symptoms four weeks later; on the other hand, we have *Bibb's* case from Mexico in which twenty or

more years elapsed between possible infection and the appearance of the clinical symptoms.

Prodromal symptoms may occur for a year or more before the outbreak.

Recurring febrile attacks are almost invariable. Unusual debility and somnolence are often present, accompanied by dyspepsia. Epistaxis, headache, hyperæsthesia, and hyperidrosis are all frequently seen as prodromata.

After a longer or shorter time a skin eruption occurs, taking the form of maculæ—either erythematous, pigmented, or vitiliginous.

These patches, often evanescent at first, soon become permanent and anæsthetic. They are generally seen first on the face, extensor surfaces of the limbs, backs of the hands, back, abdomen, and chest; and the hair is lost in the affected regions.

Following this condition, the disease develops in one or other of the two definite clinical types:—

I. **Lepra tuberosa.**—The erythematous patches, which previously showed no thickening, now begin to develop into nodules, firm, hard, and either hemispherical or oblong, or merely a large infiltrated patch.

The condition of the face is usually characteristic. The eyebrows are the seat of infiltration and nodules causing prominence, furrowing, and loss of hair. The cheeks and chin are infiltrated and deeply furrowed; and the lobules of the ears are almost always affected. This deformity of the whole countenance is generally called the *Facies leonina.*

In this Lepra tuberosa *the eyes* are nearly always affected. Nodules develop in both eyelids close to the margin. The edge of the cornea becomes cloudy, then infiltrated until its dimensions may prevent closure of the lids. Iritis is a usual accompaniment, and the ciliary nerves, as well as the choroid and retina, become infiltrated.

Of the *mucous membranes*, those of the nose, mouth, larynx, and pharynx are very frequently affected.

A general infiltration of the anterior part of the nasal mucous membrane will lead to softening and ulceration until all the soft parts have disappeared, but (unlike syphilis) the bones are never affected.

The tongue, gums, and uvula may either be covered with nodules or else infiltrated. The pharynx and epiglottis are infiltrated; the voice is rough and hoarse from involvement of the glottis. Later ulceration causes cohesion and contraction of the vocal cords, till the rima is almost obliterated, and tracheotomy may be necessary.

The *lymphatic glands* in affected areas always show an indolent swelling which never suppurates.

In addition to the above skin lesions many nerves may be involved, and always the following:—facial, radial, ulnar, median, and peroneal. The involvement is throughout the whole length, but only severe at superficial sites. The result is—severe and persistent pain, followed later by anæsthesia.

In Lepra tuberosa, the testicle, liver, and spleen are always affected.

The nodules may either remain stationary for some years or else proliferate considerably; but eventually they usually soften about the middle of their base, and the nodule sinks in over the softened part; or, in other cases, they burst, leading to ulceration, and finally leaving sunken stellate scars.

Apparently the bacilli in different cases are of varying virulence. Sometimes there are several eruptions a year; at other times there may be only one or two fresh outbursts during the whole course of the disease.

In these latter cases the prognosis is more favourable, and life may be prolonged for many years, or the disease even, in some cases, be arrested. A case of complete recovery is reported by *v. Neumann* of Vienna in 1906.

The leprosy itself influences but little the health of the patient. The nodular form indeed shortens life, but this is due to the internal amyloid degenerations which follow repeated ulceration; or, in many other cases, to a nephritis, which is very common in this type of the disease.

The *duration of life* in the nodular form averages eight to nine years after the definite outbreak of the disease.

The most frequent *complication* is tuberculosis.

It has been said that the palms and scalp are never affected in nodular leprosy. *Leloir*, however, has seen the palms affected; and *Pernet* reported two cases of scalp involvement. One, an Indian male, from Bombay in 1898, with advanced nodular leprosy, showed infiltration of part of the scalp with diffuse irregular alopecia. The second case was that of a white woman seen by *Crocker* during the early stages in 1895. In 1904, when the disease was far advanced, *Pernet* found the region near the vertex to be markedly infiltrated, with considerable loss of hair.

II. **Lepra maculo-anæsthetica.**—After the prodromal symptoms previously described (which are common to both types), if the disease is about to assume the maculo-anæsthetic rather than the nodular type, very frequently bullæ (Pemphigus leprosus) may develop on the hands, legs, or elsewhere, varying in size from that of a pea to that of an egg. These bullæ may burst, and either leave a pale patch with a pigmented border, or, more rarely, lead to the formation of a serpiginous sore.

The prodromal skin lesions may now increase, and gradually it becomes obvious that there is profound implication of the nervous system. Severe neuralgias, hyperæsthesia, anæsthesia, and paræsthesia show themselves in various parts of the body.

The implicated nerves are thickened and tender, and the condition soon leads to paresis and muscular atrophy. This is most marked in the hands and fore-arms, feet and calves, and on the face. The interossei of the hands and feet also atrophy, as well as the thenar and hypothenar muscles, giving rise to a "main-en-griffe."

In the face all the muscles atrophy, and expression is lost.

The *Orbicularis oris* being paralysed, the under lip drops, and the

same condition of the *Orbicularis palpebrarum* prevents closure of the eyes. The various atrophies are not necessarily symmetrical.

The nerve-trunk destruction leads to trophic affections of the bones and skin. Necrosis of feet and hands, with exfoliation of the bone, takes place. The fingers and toes disappear.

Trophic pressure ulcers on the sole of the foot are frequently seen. In these ways great mutilation takes place, and scarcely anything at all of the foot may be left.

In the later stages, with pronounced facial paralysis, sense of smell and taste may be diminished or lost.

The course of this type of leprosy is essentially chronic, and may persist for ten, twenty, or even forty years. Death usually takes place from intercurrent disease.

Most maculo-anæsthetic patients eventually become purely anæsthetic—that is, they no longer suffer from leprosy, but only from its results. The occasional appearance of late eruptions, however, show that bacilli may remain somewhere in the body.

Differential Diagnosis.

	Leprosy.	Tubercle.	Syphilis.
Arrangement of bacilli,	In masses.	Singly.	...
Vascularity, .	Rich in vessels.	Avascular.	Normal.
Caseous degeneration,	Never found.	Present.	...
Giant cells, .	Only multinuclear never giant cells.	Always found.	...
Heredity, . .	Only predisposition inherited.	Only predisposition inherited.	Congenital or acquired.
Involvement of:			
Brain, . .	No.	Yes.	Yes.
Meninges, .	No.	Yes.	Yes.
Spinal cord, .	Very rare.	Yes.	Yes.
Lung, . .	Very rare.	Common.	No.
Serous membranes,	Late.	Early.	No.
Skin, . .	Early and characteristic.	Uncommon	Primary exanthem and distribution quite different from leprosy.
Peripheral nerves	Yes.	Rare.	No.
Hair, . .	Yes.	No.	Rare.
Ulcerations, .	Superficial and irregular and common.	True skin ulcer very rare.	Punched out and deep.

To face p. 250.

Fig. 73. Nodular leprosy in a Malay woman. (Note the mutilation of hands.)

Pathology and Morbid Anatomy—*The Leproma.*—

1. Smooth, white, and glistening on section.
2. Nucleated cells, size of leucocytes (lepra cells).
3. A few large epithelioid cells.
4. Fragments of connective tissue and blood-vessels.
5. Bacilli both in cells and free.
6. Sweat glands, sebaceous glands, and hairs are compressed by infiltration, and finally disappear.
7. Corium becomes fibroid.

Mucous Membranes.—Here the nodules are softer and ulcerate more readily.

In the *tongue*, if the lesion is *nodular*, the growth will be chiefly composed of embryonic cells, very few lepra cells, and but slight vascularity. The growth extends to the muscles, the fibres of which are gradually destroyed. There is fibroid formation. If the lesion, on the other hand, is *infiltrated*, the neoplasm almost entirely consists of lepra cells, and there is little tendency to fibrous formation.

In the *pharynx* and *larynx* the ligaments, muscles, and cartilages become infiltrated with new growth, leading to ulceration.

Nerves.—Those most frequently affected are the median, radial, ulnar, posterior tibial, peroneal, and facial. The nerves exhibit a swelling due to infiltration of the perineurium by lepra cells. They later invade the endoneurium, giving rise to neuritis and degeneration.

Lymphatic Glands.—These may be much enlarged, especially in nodular cases. The adenoid tissue is replaced by lepra cells. In old cases there is sclerosis. Caseation and suppuration never take place unless from concurrent tuberculosis (*Hansen*).

Anæsthetic Macules.—These, like the nodules, are leprous infiltrations of the cutis, which appear to proceed from the vessels.

Bacilli are always present, and are most numerous in the younger macules. The anæsthesia is the result of atrophy of the nerves caused by secondary shrinking after the leprous affection has disappeared.

The Testes.—These are frequently the seat of leprous deposit, and, though often not enlarged, will be tough and hard to cut, due to sclerosis.

The Liver.—This organ does not, as a rule, show any macroscopic changes, but there is a diffuse infiltration of interlobular connective tissue with lepra cells.

The Spleen.—This organ is not enlarged, but innumerable bacilli-bearing cells are found.

It is doubtful if the *spinal cord* is ever directly implicated by the leprous processes.

Other organs show no special lesions.

Treatment.—The first indication is to place the patient in clean, well-ventilated, healthy surroundings.

Personal linen and bed-linen should be changed daily and soaked

in a saturated thymol solution. The same solution should be sprayed on the floors twice daily.

Abundance of fresh air, sufficient exercise, and plentiful nutritious food are indicated.

Hot baths, containing a handful of sodium chloride, should be given for five minutes twice daily.

The following or similar tonic should be given three times daily after food:—

℞.—Ferri et ammon. cit., ...	gr. x.
Liq. arsenicalis, ...	♏v.
Aq. ad	℥i.

M. Ft. Mist.

In addition to the above general treatment, the following special treatment should be tried:—

Chaulmoogra Oil.—This is the expressed oil from the nut of *Gynocardia odorata.*

The treatment consists in rubbing the oil into the skin of the affected areas, twice daily, for fifteen or twenty minutes at a time.

It should also be given by the mouth in 10-minim capsules three times daily, gradually increasing the dose until at least 2 drachms are taken daily.

The treatment should be persisted in, honestly, for several years—if necessary, six or seven—and in most cases a vast improvement is assured, and even a cure not unreasonably expected if the treatment has been begun sufficiently early.

The treatment of ulcers, &c., will naturally be on surgical lines:—

Ulcers should be scraped with a Volkmann's spoon and dressed antiseptically.

Useless members may be amputated, and should be if gangrenous.

Neuralgia, if intense, may often be alleviated by nerve stretching.

Perforating ulcers of the foot should be freely opened up and allowed to granulate.

Necrosed bone should be removed.

Cutaneous nodules in awkward situations, such as on the eyelids, prepuce, &c., may be excised.

Corneal invasion may demand an iridectomy (before the iris becomes adherent) and may preserve the vision for some time.

Laryngeal obstruction will call for tracheotomy, and a tube may be worn for many years.

In conclusion, it will be well to mention some of the multifarious drugs which have been advocated from time to time:—

Gurgun Oil (from *Dipterocarpus turbinatus*).—Used as in case of Chaulmoogra oil, but with less good results.

Mercury.—Unless complicated by syphilis this drug is useless. Intramuscular injections of the perchloride have occasionally been productive of benefit.

Salicylate of Soda.—Recommended by *Danielssen.* Of doubtful value.

Salol.—20 to 30 grs., t d s. (*Lutz*). There is probably no known disease for which this drug has not been advocated at some time or another. Where an intestinal antiseptic is indicated it is of much value.

Pot. Chlorate.—80 to 100 grs. per diem. Of doubtful value.

Tuberculin —Reaction produced, but it does not stay the disease

Thyroid Gland.—Has been tried, as have so many drugs, in Trinidad. Of doubtful value.

Leprolin.—*Rost** states that he has succeeded in growing the *B. lepræ* on a salt-free medium. After six weeks the culture medium is filtered and glycerin added. This constitutes the *Leprolin.* Injections of this cause a rise of temperature and inflammation of the lepromata. Sod chloride is administered internally, and also externally as an ointment. Very good results are said to have been attained by him, as also by *Wood, Fleming*, and others in India.

* *Ind. Med. Gaz.*, 1904. pp. 107 and 203.

CHAPTER XXVIII.

MALARIA.

(Mal'aria = bad air.)

Definition.—A specific infectious disease, due to invasion of the blood by three species of hæmosporidia of the genus *Hæmamœba*, giving rise to three different types of febrile paroxysms, all of which are amenable to quinine.

Synonyms.—*Ague; Intermittent fever; Paludism; cold, marsh, swamp, climatic, jungle, bile, mountain, coast, or gnat fever; Hæmamœbiasis* (Roos); French = *Paludisme;* German = *Wechselfieber;* Italian = *Paludismo.*

History and Geographical Distribution.—The disease has been more or less familiar to the medical world for many centuries. Certain passages in the writings of Ancient Egypt are thought to refer undoubtedly to this malady. Coming to more recent times, *Hippocrates*, in B.C. 400, discourses on tertian and quartan fevers, and even recognises a malarial cachexia.

In the first century of the Christian Era, *Celsus* likewise remarks on the periodicity of the fever.

The next step in our knowledge of malaria came in 1640, when the Viceroy of Peru—Del Cinchon—and his medical adviser, *Del Vego*, brought to Spain some samples of the bark which we now know as Cinchona, and which the latter was the first to reeognise as having a specific action in the treatment of malaria.

In 1712 *Torti's* classical medical work appeared, with descriptions of pernicious malaria.

At the beginning of the nineteenth century *Andouara* called attention to the splenic enlargement in malaria.

In 1850 *Virchow* remarked on the accompanying melanœmia.

Finally, in 1880, *Laveran*, in Algiers, discovered the specific parasite, at first disputed, but soon amply confirmed.

That the disease might be conveyed by mosquitoes was suggested by *King* in 1883, *Laveran* in 1891, *Manson* in 1894, and *Bignami* in 1896

The demonstration of the correctness of this theory was proved by *Ross* (1897-9), who traced the development of the parasite in the mosquito. The whole subject has been admirably worked out by the Italian School—*Grassi*, *Celli*, *Feletti*, *Bignami*, and others.

The distribution of malaria is widespread through temperate, subtropical, and tropical countries. It is rarely, if ever, met with above the latitude of 60° N., and, as the tropics are approached,

PROTOZOA.

Classes.—Rhizopoda. Flagellata. Sporozoa. Infusoria.

Orders:—Gregarinida. Coccidiida. Hæmosporidia. Mixosporidia. Microsporidia. Sarcosporidia.

Genera: * Hæmamœba. Hæmogregarina. Piroplasma. Halteridium.

Species. *H. vivax* (Benign tertian). *H. malariæ* (Quartan) *H. præcox* (Malign. tertian).

* Of this genus there are, in addition to the three human species here given, other species from monkeys, birds, bats, and reptiles, so that malaria is not confined to man.

endemic malignant malaria is found, as well as the benign types which alone prevail in temperate regions.

Australia and most of the Pacific Islands are exempt. In Japan it is comparatively rare.

With these exceptions, malaria is met with throughout most of the tropics and subtropics, especially near lakes, rivers, and marshes.

In many parts of Europe it is frequently found.

Etiology.—The parasites which cause malaria, and which, as we have already seen, were discovered by *Laveran* in 1880, belong to the genus *Hæmamœba* (*Grassi*). The alternative, and perhaps most popular, name for this genus is "Plasmodium," but, as *Scheube* and *Braun* have pointed out, this is a most inappropriate name, which implies not a separate creature, but congeries arising from the coalescence of single-celled organisms. The older and more appropriate term of Grassi—*Hæmamœba*—is, therefore, here given to the genus. The place of these parasites in the zoological system is as on p. 261.

Before discussing the malarial parasite and its life history, it will be well to give a short glossary of terms used in this zoological connection, for the nomenclature is now so varied that it is often hard to understand what is meant by a given term. Observers, alas, too often indulge in the habit of coining their own terms, and the endless confusion that results is not only perplexing to a learner, but a subject for ridicule to the scientific world. *Quot auctores tot nomina!*

Malarial Nomenclature.

Ague = intermittent malarial fever.

Amphigony (αμφιγονος, having two parents) = sporogony (*q.v.*).

Benign tertian, type of fever caused by *H. vivax*.

Blastophore (βλαστος, a bud; φερειν, to bear) = sporoblast (*q.v.*).

Chromidia = nucleolar elements of a male gamete.

Endemic index = the percentage of infected children (under ten) in any district. It represents the liability of immigrants to contract malaria.

Endogenous cycle, or schizogony, being the developmental cycle of the malarial parasite within the human host.

Exogenous cycle, or sporogony, being the developmental cycle of the malarial parasite, partly in the human host, and partly in an intermediate host.

Gamete (γαμος, union) = sporont (*q.v.*).

Gametocyte = sporont (*q.v.*).

Germinal rods = sporozoites (*q.v.*).

Gymnospore (γυμνος, naked) = sporozoite (*q.v.*).

Karyosome (καρυον, kernel) = remains of a sporont nucleus.

Laverania malariæ (Grassi) = *H. præcox*, the parasite of malignant malaria.

Macrogamete = female sporont which has eliminated some of the nuclear substance.

Malarial endemicity = endemic index (*q.v.*).

Melanin (μελας, black) = pigment formed by the malarial parasite, possibly altered hæmatoidin.

Merozoïtes (μερος, a part; ζωον, animal) = round spores formed in the endogenous cycle.

Microgametes = flagella extended by male sporonts.

Monogony (μονογονος, having one parent) = schizogony (*q.v.*).

Myxopods, μυξα, mucus; πους, a foot) = merozoites (*q.v.*).

Oökinete (ωον, an egg; κινησις, movement) = vermicule (*q.v.*), or travelling vermicule.

Parthenogenesis, as applied to the malarial parasite, signifies the multiplication of the resistant female sporont (if not removed from the body) to form fresh schizogonous generations, manifested clinically by relapses.

Plasmodium falciparum (Blanchard) = *H. præcox*, the parasite of malignant malaria.

Plasmodium immaculatum (Schaudinn) = *H. præcox*, the parasite of malignant malaria.

Plasmodium malariæ (Schaudinn) = *H. malariæ*, the quartan parasite.

Plasmodium vivax (Schaudinn) = *H. vivax*, the parasite of benign tertian malaria.

Quartan fever = malarial fever of 72 hours' cycle, caused by *H. malariæ*.

Quotidian ague = daily malarial fevers, caused by the sporulation in multiple infections.

Schizogony (σχιζω, to cleave) = the endogenous life cycle.

Schizont = the mature, non-amœboid, spore-forming parasite, which produces the endogenous reproduction.

Spores—(*a*) In the endogenous cycle are round and called merozoites. (*b*) In the exogenous cycle are spindle-shaped and called sporozoites.

Sporoblast = the third stage of the zygote, consisting of spores formed round the divided nuclei (or synkaryon).

Sporogony = the exogenous life cycle of the malarial parasite.

Sporont = the mature, sexually differentiated parasite, concerned in exogenous reproduction.

Sporozoites = spindle-shaped spores formed during the exogenous cycle.

Synkaryon (συν, together; καρυον, a nut, a kernel) = the nucleus of a vermicule.

Trophozoites = the minature amœboid parasites in corpuscles.

Vermicule (or "travelling vermicule") is the name given to a zygote during its second stage, when it becomes elongated and motile.

Zygospore = zygote (*q.v.*).

Zygote.—The sexual result of fertilisation of a macrogamete by a microgamete.

Zygotoblast = sporozoite (*q.v.*).

Zygotomere = oökinete (*q.v.*).

Life History of the Malarial Parasite (Fig. 54).—The malarial parasite has two methods of reproduction. The one is an *endogenous reproduction*, or *schizogony*—so-called because the whole process takes place within the body of the host by division into a number of spores, each of which can attack and enter a fresh blood-corpuscle.

The other method of reproduction (or *exogenous reproduction*) is

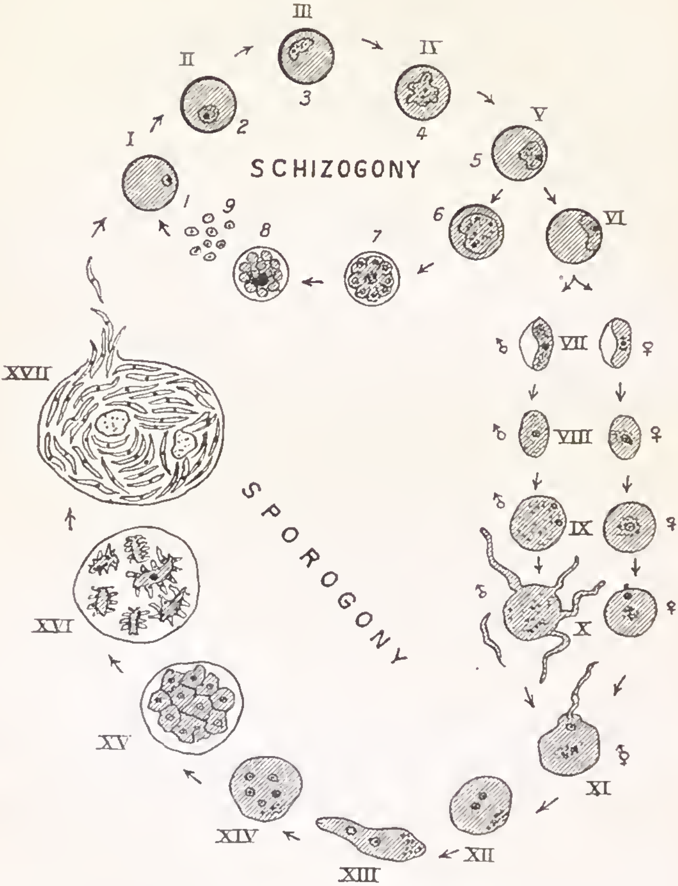


Fig. 54.—The Malarial Parasite. Diagram of the Double Cycle.

a sexual one, in which fertilisation takes place with the ultimate production of spindle-shaped spores, which, in their turn, can each infect a new red corpuscle. This method is called "*sporogony*," and for its completion the assistance of some intermediate host, such as the mosquito, is necessary.

This provision, of course, ensures the survival of the parasite.

To commence with **schizogony**:—Spindle-shaped spores (or sporozoites) are introduced into the blood by the proboscis of a mosquito. These each penetrate a red corpuscle, becoming an amœboid trophozoite which commences to grow within the corpuscle, thus beginning the endogenous cycle.

The main scheme of this, as well as of the exogenous cycle, is the same for all the three species of malarial parasites.

The morphological points which differentiate them will be found in a tabular statement subjoined.

In the early stages the young trophozoites assume a form much like that of a signet ring. This is considered by *Schaudinn* to be due to the formation of a vacuole near the nucleus, possibly in order to increase the absorption surface of the parasite.

As the parasite grows the vacuole disappears, and the black grains of melanin, formed by the parasite, become obvious.

When full grown the parasite practically fills the corpuscle, is no longer amœboid, and is known as a *schizont*.

The pigment collects in the centre, the nucleus divides into a number of parts, the protoplasm becomes segmented, and, finally, a number of round spores (*merozoites*) are matured and break away, each to attack a fresh red corpuscle, and so renew the endogenous cycle.

Sporogony, or the Exogenous Cycle.—The rapid increase by geometrical progression, as detailed above, soon leads to a reaction in the body of the human host, and fever results.

This reaction is considered by *Schaudinn* to act as a stimulus for the production of the sexual sporonts, which carry on the exogenous reproduction.

Whether or not this is the case is uncertain, but a certain number of the amœboid trophozoites, instead of growing into schizonts, become sexual *sporonts* (or gametocytes). In these cases the ring form does not occur, and the growth is much slower than in those parasites destined to become schizonts. When the parasite has reached a certain size, instead of nuclear division and protoplasmic segmentation, male and female sporonts are formed. In the case of malignant malaria the sporonts are sausage or crescent shaped, but in the benign tertian and quartan they are irregularly round or oval. In the male the nucleus is central and the pigment granules scattered; in the female the nucleus is also central, but the pigment is aggregated around it.

These full-grown sporonts circulate in the peripheral blood, al-

though in the early stages (unlike the young schizonts) they are, as a rule, only to be found in the spleen and bone marrow.

At this stage, if not taken up by a mosquito, the male sporonts appear to die off; but the females are more resistant, and may remain dormant in some internal organs after the death of all the other parasites, being then capable of parthenogenesis and of restarting a schizogonous cycle — manifested clinically as a relapse.

If, on the other hand, the adult sporonts happen to be ingested by a mosquito, they pass into the mosquito's stomach with the human blood. In the male sporonts the nucleus gives off *chromidia*, which travel to the surface of the parasite and are extruded, with some of the protoplasm, in the form of some five or six *flagella* (*microgametes*). These become detached and seek the female sporont (*macrogametes*), which they enter, and with the nucleus of which their nucleus is fused, thus forming a fertilised parasite or *zygote* (see Figs. xi. and xii. of Fig. 54). This zygote becomes elongated and mobile, and is then known as a *vermicule* or *oökinete* (see Fig. xiii. of Fig. 54). Its anterior extremity is rather sharp, the nucleus is behind the centre of the body, the pigment is massed at the hinder end. With active movements the vermicule pierces, and is arrested just under, the epithelium of the mosquito's stomach. Here it becomes spherical and encysted, and by absorbing nourishment from its host increases in size, forming slight projections into the lumen of the stomach.

The nucleus (*synkaryon*) multiplies by division, and round these daughter nuclei, the protoplasm is aggregated, forming *sporoblasts* (*vide* Figs. xiv. and xv. of Fig. 54).

The nucleus of each sporoblast further divides, and these smaller nuclei travel to the surface and become enclosed in slight protoplasmic protrusions known as *gymnospores* (*vide* Fig. xvi. of Fig. 54).

These protrusions become detached and fill the cyst, to the number of some hundreds or thousands of spindle-shaped spores, known as *sporozoites* (*vide* Fig. xvii. of Fig. 54).

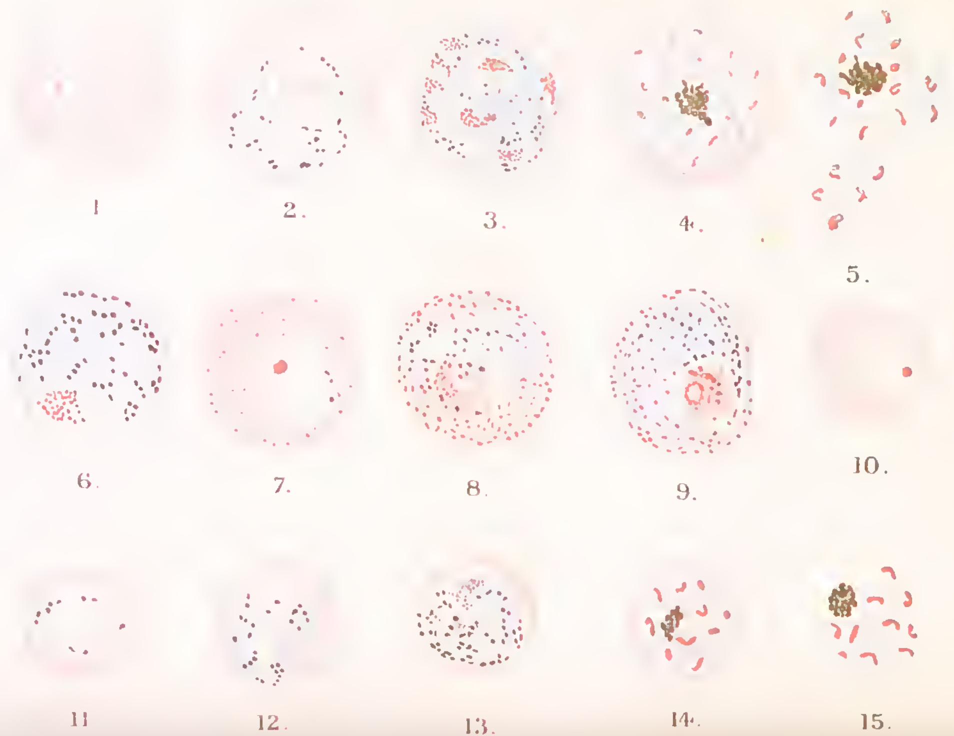
When ripe these cysts rupture, and the sporozoites become free in the tissues of their mosquito host. They pass along in the blood fluid, and, being actively mobile, eventually reach the salivary glands.

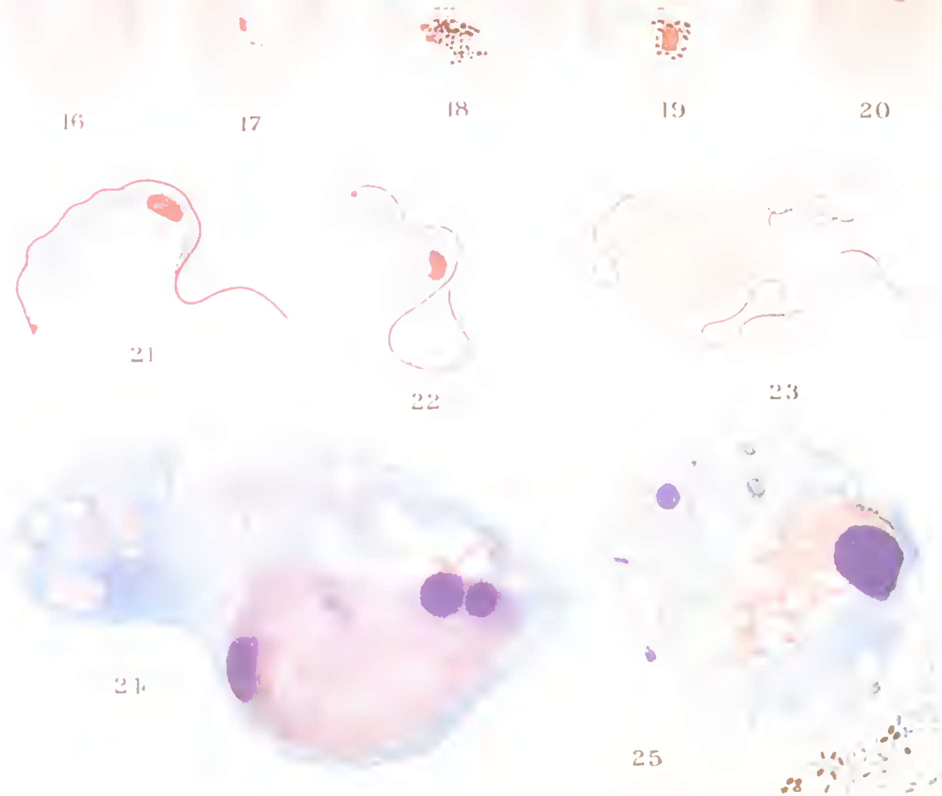
When the mosquito next feeds, these sporozoites pass down the proboscis and are injected into the wound, thus reaching the blood stream of a fresh human host. Each sporozoite can attack and enter a red blood-corpuscle in the same way that the merozoites do, thus completing the exogenous cycle, which, in the body of the mosquito, lasts for about ten or twelve days.

Whether other insects than mosquitoes can act as intermediate hosts, or whether the parasite can live in vegetable juices and be thence obtained by mosquitoes, has not yet been proved.

Definite proof has been obtained, however, that certain of the

MALARIAL & OTHER PARASITES





16 17 18 19 20 21 22 23 24 25

A. [illegible]

Bale & Danielsson, L.td lith

Anophelinæ can act as efficient hosts. Of the 12 genera of the *Anophelinæ* over 80 species are known, but only the following have been definitely incriminated :—

Mosquito Hosts of the Human Malarial Parasite.

Europe.

Anopheles maculipennis.
Anopheles bifurcatus.
Pyretophorus superpictus.
Myzorhynchus pseudopictus.

North America.

Anopheles maculipennis.

Japan.

Anopheles jesoensis.

Central and South America.

Nyssorhynchus lutzi.
Nyssorhynchus cubensis.

Africa.

Myzomyia hispaniola.
Myzomyia funesta.
Pyretophorus superpictus.
Pyretophorus costalis.
Anopheles maculipennis.
Anopheles algeriensis.
Myzorhynchus paludis.

West Indies.

Cellia albipes.

India.

Myzomyia listoni.
Myzomyia culicifacies.
Nyssorhynchus maculatus.

In addition to the above, the malarial parasite of birds—*Plasmodium præcox*—is conveyed by *Culex pipiens.* That of monkeys—*Plasmodium kochi*—by a species of mosquito as yet undiscovered; nor has the malaria of bats yet been worked out.

For methods of making and staining blood-films see Chapter xlii. For coloured reproduction of the parasite see annexed Plate.

Stained with Leishman's Stain.

FIGS.

1 to 5. Stages of benign tertian parasite.
6. Gamete benign tertian.
7, 8, 9. Characteristic degeneration of red corpuscles containing benign tertian parasites (Schüffner's dots).
10 to 15. Stages of quartan parasite.
16, 17. Stages of malignant tertian (sub-tertian) which are seen in peripheral blood.
18. Male gamete, malignant tertian (sub-tertian).
19. Female gamete, malignant tertian (sub-tertian).
20. Double infection with malignant tertian (sub-tertian) parasites of a red corpuscle; basophilic granules in red corpuscle.
21. *Trypanosoma lewisii* (rat).
22. *Trypanosoma gambiense* (Congo).
23. Spirillum of relapsing fever (stained with carbol fuchsin).
24, 25. *Amœba coli.*

The following are the morphological differences between the three malarial parasites :—

	Tertian.	Quartan.	Malignant.
Time of schizogonous cycle,	48 hours.	72 hours.	Irregular.
Pyrexia resulting,	Every other day.	Misses 2 days.	Irregular or continued.
Effect on corpuscles,	Enlarged and pale.	Diminished or normal, and normal colour.	Not definite. Parasite small.
Pigment, . .	Fine. Considerable.	Coarse.	Scanty.
Amœboid tendency,	Very marked.	Less marked.	Active.
No of merozoites,	12 to 24.	9 to 12.	Variable, but few.
Site of sporulation,	Peripheral blood.	Peripheral blood.	Liver, spleen, and bone marrow.
Male sporonts, *	Circular or oval. 10 μ in diameter. Large nucleus central. Lightly-stained protoplasm.	Circular or oval.	Crescent or sausage-shaped. Pigment granules scattered.
Female sporonts,*	Circular or oval. 12-16 μ in diameter Cytoplasm stains deeply.	Circular or oval.	Crescent or sausage-shaped. Pigment granules aggregated round nucleus.
Schüffner's dots (*i.e.*, fine red granules throughout infected corpuscle),	Present.	Absent.	Absent. Coarse granules and clefts may occur round parasite.

* It is only at maturity that the sporonts are seen in the peripheral blood.

Experimental Proof of the Mosquito-malarial Theory.—Experiments on behalf of the Colonial Office and of the London School of Tropical Medicine were carried out in 1900.

Drs. *Sambon* and *Low*, with visitors and servants, lived for the three most malarial months of the year in Ostia (one of the most malarial localities of the Roman Campagna). They moved about freely during the day in all weathers, drank the local water, took no quinine—in fact, took no precautions whatever beyond retiring to their mosquito-proof hut from sunset to sunrise. The neighbouring Italian peasants all suffered from malaria, while the experimental party enjoyed complete immunity.

While this experiment was in progress, some mosquitoes were fed in Rome on the blood of malarial patients, and subsequently forwarded alive in suitable cages to the London School of Tropical Medicine, where they were allowed to feed on the late Dr. P. Thurburn Manson and George Warren—the laboratory assistant—neither of whom had been abroad or exposed to any malarial influences. They both developed fever in about fourteen days, and abundant malarial parasites were found in their blood.

Requisite Conditions for the Appearance of Malaria.—As far as we at present know, all of the following factors must be present:—

1. The presence of definite genera and species of the *Anophelinæ*.
2. The present or recent existence of human malarial cases.
3. Favourable climatic conditions for the mosquito.
4. Susceptibility of the mosquito.
5. Susceptibility of the healthy person bitten by the infected mosquito.
6. An interval of some ten or twelve days between the time that the mosquito has fed on infected blood and the biting of a healthy person.

Immunity.—That a certain amount of immunity may be acquired is considered by some as most probable, on account of (1) the relative insusceptibility of certain natives; (2) the insusceptibility of certain individuals to repeated exposure or experimental inoculation.

A. Plehn, however, considers that one attack predisposes to others—possibly relapses; but that relapses, if properly treated, become less severe until finally tolerance is established, which is not absolute, nor is it a true immunity. Removal to another neighbourhood, or lessened resistance, may result at any time in increased virulence on the part of the parasite.

The hypothetical endotoxins arising from the dead protoplasm at the time of sporulation, seem to disappear quickly, and thus allow any remaining parasites to continue their development until the next sporulation.

There is no reactive formation of antibodies, and no permanent active immunity.

Among natives a certain type of immunity seems to be often inborn, and children's blood may exhibit the parasite without signs

of fever. This condition probably begins during fœtal life, when the endotoxins circulating in the maternal blood pass into the placenta circulation.

Europeans may acquire a somewhat similar immunity by the prophylactic use of quinine, for the parasites are accessible to the action of quinine at some stage, and the latency is produced when the parasites have been destroyed by the quinine and the endotoxins liberated. To secure the action, half a gramme of quinine should be administered every fourth day while the individual is residing in a malarial neighbourhood, and for six months after leaving it, or after the last relapse.

Predisposing Causes—1. *Climate and Season.*—A certain amount of heat and moisture are necessary for the development of the *anophelinæ.*

In the tropics these conditions are, of course, obtained, although the wet season will be the most suitable time for the mosquitoes to breed.

In temperate climates the disease only appears during the warmer months of the year, and of these the late summer or early autumn are most favourable to the spread of the malignant variety; hence the name sometimes given to it, "æstivo—autumnal fever."

2. *Moisture.*—Low and marshy land is especially malarious, since it is admirably adapted for breeding grounds.

3. *Soil.*—Impervious subsoil giving rise to swampy collections of water is highly dangerous, being especially favourable for breeding of *anophelinæ.*

The higher this subsoil water the greater the chance of stray pools.

Railway cuttings, embankments, &c., during construction are frequently the cause of a rise in the subsoil water. It is well known that gangs of labourers employed in such undertakings are visited by outbreaks of malaria, which are usually attributed to some inherent malarial property of the newly-turned soil. It has, however, never yet been proved that no malarial focus was present amongst the labourers themselves, nor that *anophelinæ* were absent. Until this is done, therefore, it is not unreasonable to conclude that the engineering undertakings, by raising the neighbouring subsoil water and by creating surface holes, have given the best facilities for the propagation of any *anophelinæ* in the neighbourhood, and that such mosquitoes have found an infected workman. An outbreak will naturally follow.

4. *Food or Water.*—There is no evidence that the malarial parasite can live in, or be conveyed by, water or food of any kind.

5. *Age, Sex, and Race.*—Children and infants are more susceptible than adults, so much so that *Koch* considers the percentage of infected children under ten to be an index to the amount of malaria in that locality, and to represent the liability of immigrants to contract malaria (endemic index). Certain natives of malarious tropical regions seem to be partially immune. This has been ascribed to frequent infections during youth.

6. *Locality.*—The prevalence of malaria is limited to the geographical range of the incriminated *anophelinæ*, and is therefore rare at most high altitudes.

A person once infected may, however, suffer from relapses, although far from the locality of incidence. This fact should be borne in mind when investigating cases, in which otherwise an error might easily be made.

Pathological Anatomy—1. *The Blood.*—There is a tripartite anæmia. The volume of the blood is diminished, there is a diminution of the hæmoglobin value, and there is an oligocythæmia.

A characteristic leucocytic variation also occurs as a relative increase in large mononuclear elements, which may reach 20 per cent. or more of all leucocytes found. This condition may be found as long as three or more months after an attack.

The methods of taking and staining blood films is dealt with in Chapter xlii. (*q.v.*):—

In unstained specimens—

$\left.\begin{array}{l}(a)\ \text{Concave mirror}\\(b)\ \text{Small stop}\\(c)\ \text{No condenser}\end{array}\right\}$ should be used.

In stained specimens—

$\left.\begin{array}{l}(a)\ \text{Flat mirror}\\(b)\ \text{Large stop}\\(c)\ \text{Condenser}\end{array}\right\}$ should be used.

2. *The Spleen.*—In this, as in the other organs, the conditions described refer chiefly to the malignant variety, since death from benign infections is rare.

The spleen is enlarged, soft, and dark or black. The pulp is crowded with infected corpuscles. There is marked phagocytosis, some of the phagocytes containing entire blood-corpuscles as well as parasites and pigment.

On section, the surface is of a slaty colour and the trabeculæ prominent.

In old-standing cases amyloid changes may be present.

3. *The Liver.*—This organ is dark-brown or slate-grey in colour, and is usually enlarged. In chronic cases there may be a non-atropic perilobular fibrosis, and amyloid changes occasionally supervene. The preliminary hepatitis rarely, if ever, proceeds to abscess formation.

4. *The Brain.*—Punctiform hæmorrhages are frequently found. The capillary endothelium is phagocytic and swollen, and the lumen will be found to be crowded with infected corpuscles. The grey matter is darkened.

5. *The Bone-marrow.*—This is much pigmented, the pigment being found not only in the blood-vessels, but, as in the case of the spleen, in the parenchymatous cells also.

6. *The Kidneys.*—There is slight enlargement, with a pale cortex, and little or no pigmentation.

Pigmented leucocytes and infected corpuscles may be found in the glomeruli and intertubular vessels. In very rare instances the kidneys may be the chief seat of localised infection, as in Ewing's fatal case of acute hæmorrhagic malarial nephritis.

7. *The Gastro-intestinal Tract.*—The pathological appearances are usually insignificant, although occasionally the intestine may be the seat of special infection.

In one case under the author's care, a Chinese boy was admitted to hospital with malignant malaria and continued fever. In two days the temperature dropped to sub-normal, and the stools became loose, green, and extremely offensive. This condition continued for a day and a half when death occurred. Post-mortem:—Two feet of the small intestine, just above the ileocæcal valve, were found to be practically gangrenous, with an extensive small-celled infiltration of the submucosa, some sloughing of the mucosa, and evidences of pigmentation. Whether the two conditions were in any way related is an interesting point.

Symptoms.—After a healthy individual has been bitten by an infected mosquito, the sporozoites, thus introduced, attack blood-corpuscles.

These parasites multiply by schizogony in geometrical progression, until, after a variable time, they become sufficiently numerous to cause a reaction in the body of their human host. This *incubation period* varies according to the parasite, *e.g.*—

In benign tertian (Fig. 55) it is . . .	14 days.
In quartan it is	21 ,,
In malignant fever it is . . .	10 to 12 ,,

These periods are those which have been determined by close observation or experimental inoculation, but they may apparently be prolonged in many cases.

Tertian Fever.—If a single infection has taken place febrile paroxysms will occur at intervals of about 48 hours, representing a general sporulation of the parasites in the blood.

If two different infections have occurred, sporulation of the two generations will naturally take place at different times, and a more or less quotidian fever will result.

In the same way multiple infections may give rise to an irregular kind of remittent fever.

There are seldom any prodromata.

The patient, who has been feeling quite well previously, is quickly attacked by malaise, headache, and perhaps vertigo or nausea. If the temperature is taken it will be found to be slightly raised. Very soon a feeling of chilliness supervenes, amounting in most cases to a definite rigor. The pupils are dilated; the pulse is small, of high tension, and fast; the headache is severe; and nausea and vomiting are not uncommon.

After about ten minutes to one hour, during which the temperature has been rapidly rising, the rigor passes off, and subjective

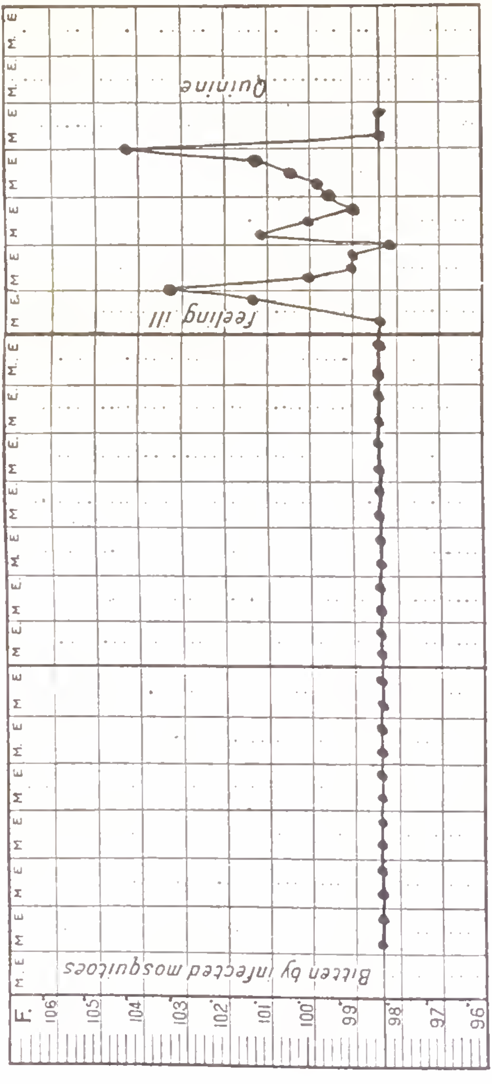


Fig. 55.—Benign-tertian incubation. (Experimental infection by mosquitoes in London, September to October, 1900.)

sensations of intense heat are experienced, generally known as the *hot stage*.

The skin is flushed, hot, and dry. The pulse is softer, and some-

times dicrotic. The initial headache persists, and there is intense thirst. The skin is sallow and yellowish; the face flushed, and

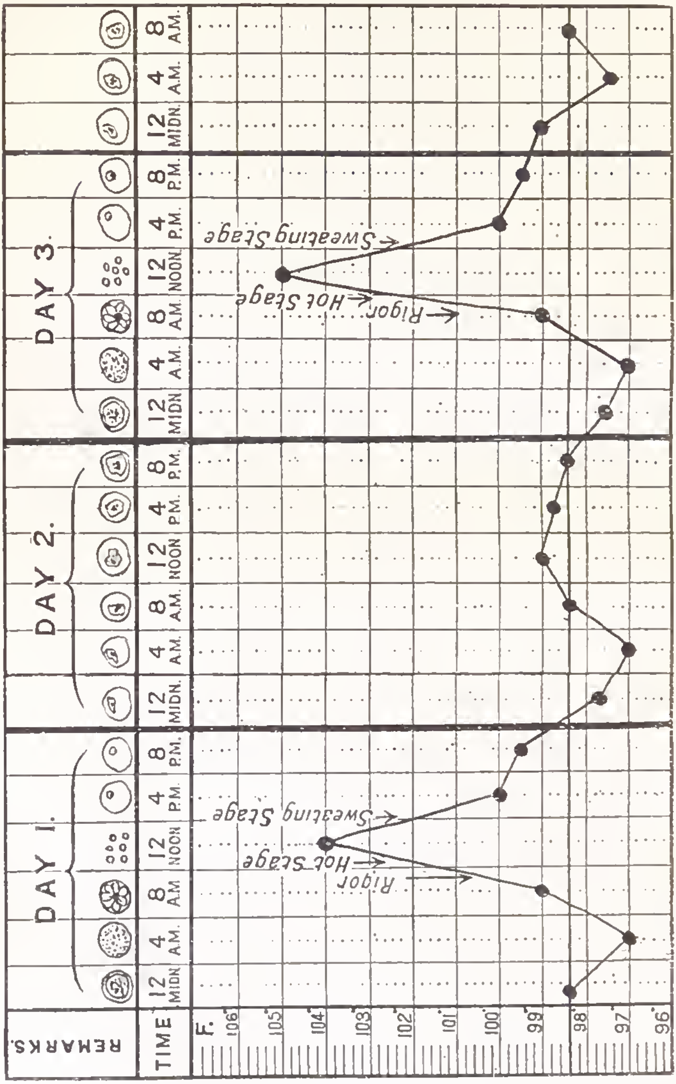


Fig. 56.—Single benign-tertian infection (showing the concurrent development of the parasite during the different clinical stages).

conjunctivæ infected; the tongue is dry and dirty brown. It is during this stage that the temperature reaches its maximum—about 104° or 105° F.

After continuing for four or five hours the hot stage terminates by diaphoresis which is often profuse. The temperature falls in a few hours to normal, or even below normal. The pulse slows and becomes more regular, and all the urgent symptoms abate until the next paroxysm.

If untreated, the paroxysms will recur with great regularity.

The usual time for attack is between midnight and midday.

The connection between the clinical symptoms and the life history of the parasite is shown in the annexed chart (Fig. 56) of a single benign-tertian infection, above which is given a diagram of the parasite at the different stages.

It will be noticed from the chart that the parasite (and this is true except during spontaneous recovery, or under the influence of

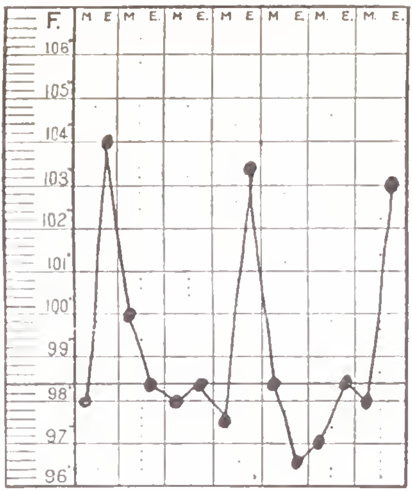


Fig. 57.—Single quartan infection.

quinine) persists in the blood during the period of afervescence, and, therefore, we gather that the fever is not directly due to the parasite. It is more than possible that when sporulation is completed, and rupture of the corpuscle has set free the merozoites, that some toxin, elaborated by the parasite, is at the same time liberated, and is the cause of the febrile attack.

Quartan Fever.—As in the case of tertian fever, there may be either single or multiple infections.

A single quartan infection (Fig. 57) gives rise to sporulation of the parasite and febrile attacks at intervals of about seventy-two hours; that is to say, that there will be a period of two days with normal temperature intervening between each febrile paroxysm.

The quartan parasite (*H. malariæ*) is seen in the peripheral cir-

culation with much greater relative frequency than are the other forms.

It is this type of disease which tends to be more common in temperate climates, and it certainly runs a more benign course, so much so that occasionally all stages may be observed in the blood of a patient free from fever, although with a tertian infection the presence of segmenting parasites almost invariably portends an approaching paroxysm.

The symptoms of an actual attack resemble those of a benign-tertian infection and need not, therefore, be again enumerated.

A cachectic state is much less easily induced in this type of malaria than in the other forms.

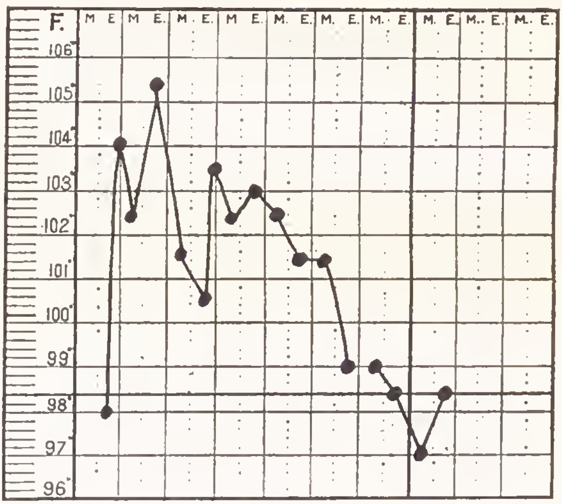


Fig. 58.—Type of malignant infection (normal course).

Malignant Malaria.—This most serious type of malaria (due to infection by *H. præcox*) only occurs in temperate climates during late summer and autumn; hence the Italian name, "æstivo-autumnal fever." In the tropics, however, it prevails fairly widely. In most ways it differs essentially from the two benign varieties.

The parasite has a 48-hour cycle, and, therefore, is frequently known as "malignant tertian." Multiple infections are, however, the rule, and the type of fever is notably irregular, the pyrexia usually being remittent or subcontinuous.

The paroxysms often extend to twenty-four hours in length. Rigors are less common than in the benign types. Pernicious symptoms are frequently manifested.

Other points of difference are—that the parasite is almost unpigmented; that, while the young forms are seen in the peripheral blood, the sporulating forms are confined to the spleen and other organs, and do not circulate; moreover, the gamete is crescent-shaped, and not round as with the benign types.

For practical clinical purposes we can divide *malignant infections* into three main types:—

1. A normal type.
2. A gastro-intestinal type.
3. A cerebral type.

1. *The Normal Type* (Fig. 58).—Most of the clinical symptoms resemble those of the benign infections, but are all more aggravated.

The headache is worse; vomiting and diarrhœa are more frequently met with, especially in children; either delirium or a pseudo-typhoid state are not uncommon. There is usually more severe fever, of a remittent rather than intermittent course.

After apparent recovery there is a considerable tendency to relapses, at intervals of eight to fourteen days.

There is often much destruction of blood-corpuscles in these fevers, and they are usually followed by serious cachexia.

During the progress of such an attack it is not uncommon for grave symptoms to develop.

2. *The Gastro-intestinal Type.*—In these cases some part or other of the gastro-intestinal tract seems to be especially involved:—

(*a*) *The Stomach.*—We then have what is known as a "bilious remittent" type.

With each fresh paroxysm there is intense gastric distress, with bilious vomiting and sometimes diarrhœa. The skin is markedly jaundiced. In such cases, although the mortality is not very great, yet profound anæmia usually results. In other cases the condition may gradually pass into a typhoid state, with low, muttering delirium and dry tongue, of which state the prognosis is bad.

(*b*) *The Intestinal Tract.*—A malarial infection in this situation may result either in a dysenteric or a choleraic type.

In the former a sudden attack of diarrhœa, with blood and mucus in the stools, may supervene, or hæmatemesis and hæmorrhages from the bowel occur without warning. In the choleraic type a sudden watery diarrhœa occurs—not the rice-water stool of cholera, as it always preserves a certain bilious character, but leading to aphonia, sunken features, shrivelled fingers, suppression of urine, and fatal collapse.

3. *The Cerebral Type.*—The following is a résumé of a description by *Thayer*:—

After several paroxysms, with the usual delirium and vomiting, drowsiness or coma supervene. The pulse becomes rapid, feeble, and irregular. The skin is hot and dry. The conjunctivæ are

injected; the tongue is dry and coated. The reflexes are often absent; the pupils dilated.

The temperature may remain high, with continued coma, for several days.

If the attack is not fatal, a gradual improvement may occur, with defervescence and sweating, during which the patient is either dull and apathetic or actively delirious.

Tetanic convulsions have been observed. Other cerebral symptoms are not infrequently seen, such as meningitis, transient hemiplegia, aphasia, amaurosis, vertigo, and some symptoms described by *Torti* and *Angelini* as resembling those of disseminated sclerosis.

It is probable that all these cerebral symptoms are due to embolism of the cerebral capillaries by the malarial parasite and its products. It was formerly thought, by those who noticed pigment in the cerebral capillaries, that the symptoms were due to a resulting thrombosis, but post-mortem observation has been credited with demonstrating the parasitic embolism.

This is almost certainly the case in those instances of *malarial amblyopia* which sometimes supervene in a cerebral pernicious attack. Since amblyopia is sometimes caused by overdoses of quinine, it is as well to copy here the differential table usually given in the text-books:—

AMBLYOPIA.

	From Quinine.	**Malarial.**
History, . .	Large doses—30 grs. or more.	Quinine may or may not have been given.
Onset, . . .	Sudden—in both eyes. Also deafness.	Not necessarily both eyes. No deafness.
Pupils, . .	Dilated. No reaction to light.	React to light.
Vision, . .	Completely lost for a time.	Never quite lost.
Ophthalmoscope,	White haze over fundus. Disc pale. Vessels constricted.	Optic neuritis. Disc grey-red. Retinal hæmorrhages.
Termination, .	Usually slight permanent defect in vision or colour.	Some cases recover completely.
Treatment, . .	Stop quinine. Give amyl nitrite.	Give Quinine.

Complications and Sequelæ.—*Pneumonia* is a frequent complication or sequel. It is, however, not of malarial origin, although the diminished resistance after malarial attacks doubtless favours the incidence.

Enteric Fever.—It was once thought that a separate entity—typho-malarial fever—existed. This is not so. Occasionally the two diseases may exist simultaneously, as in a case which *Craig* has reported. More often, however, a malarial attack will be acquired during a typhoid convalescence.

Dysentery, *cholera*, *tuberculosis*, or any of the specific infectious diseases may co-exist with malaria.

Severe *neuralgias*, *neurasthenia*, or *hysteria* may follow a malarial illness, but post-malarial psychoses are rare. The most common conclusion of a severe malaria is :—

Malarial Cachexia (*Cachexia paludéenne*).—In consequence of acute and frequent malarial attacks, a condition of anæmia is established, associated with a chronically enlarged spleen.

The skin is clay-coloured or yellowish-green, especially on the face and the dorsum of the metacarpal and phalangeal joints. There is emaciation, weakness, and lassitude. There is diaphoresis, insomnia, and anorexia.

Gradually a chronic parenchymatous nephritis is set up, marked by dropsy and albuminuria. Miscarriages often take place, and sterility is a frequent consequence of the cachexia (*Weatherley*).

In advanced cases eye affections are not uncommon, such as :—

Keratomalacia, in which both corneæ may slough in twenty-four hours. Quinine and immediate enucleation of the eye first affected should be tried.

Keratitis dendritica (Kipp), consisting of a superficial serpiginous ulceration of the cornea, accompanied by photophobia, lachrymation, and supraorbital neuralgia.

Keratitis profunda (Kipp), occurring as a grey infiltration in the middle or deep corneal layers. It should be treated by quinine internally, and atropine and fomentations locally.

Note.—*Van der Burg* and *Martin* advise the administration of quinine in every tropical corneal affection attended by fever.

Optic neuritis, *retinal hæmorrhages*, and *opacity of the vitreous* are all occasional eye developments in malarial cachexia.

Gangrene may occur in the cheeks, gums, genitals, extremities, &c. Multiple abscesses and boils are not infrequent. The cachectic condition may drag on for years, with occasional transient attacks of fever.

Death finally ensues from exhaustion, amyloid or leukæmic conditions, or from intercurrent disease.

Diagnosis.—The examination of the blood is the essential procedure in diagnosis, and it is impossible to make reliable examinations unless familiar with the appearances of normal blood. For remarks on this subject the reader is referred to Chapter xlii.

Five main diagnostic points will be at our disposal :—

1. The presence of the parasite in the corpuscles.
2. The presence of pigmented leucocytes.
3. A relative increase in large mononuclears.
4. An enlarged spleen.
5. A response to quinine treatment.

Malignant malaria is the type most likely to be confused with other conditions, such as typhoid, phœbism, uræmia, cerebral hæmorrhage, and yellow fever.

Malarial cachexia may easily be mistaken for kala-azar and leukæmia.

Prognosis.—Tertian and quartan infections practically always recover, and certainly do so when treated with quinine; quartan fever, however, is very tenacious, and relapses may occur after long intervals.

In the malignant variety the prognosis should be very guarded in untreated cases. Spontaneous recovery sometimes occurs, but there is a great tendency to cachexia. With early treatment the prognosis is quite good, but if pernicious symptoms show themselves the outlook is extremely grave.

Treatment.—Quinine is a specific. The organisms rapidly disappear after taking the drug, with the exception of the gametes, which are but little affected.

The action is especially noticeable on the very young merozoites.

The principle of quinine administration therefore is to secure its presence in solution in the blood at the time of sporulation.

Quinine is rapidly absorbed. It appears in the urine in fifteen minutes. Most of it is eliminated in twenty-four hours, but some remains, and it has therefore a cumulative action.

It was formerly thought that heroic doses of 15 to 30 grains were necessary to cure the condition; but it can be experimentally proved that 5 grains are sufficient to kill a generation of benign parasites. In malignant infections double doses can be given with advantage.

Quinine acts best when in solution, but, unfortunately, the salt most commonly sold and used is the sulphate, which is very insoluble. This difficulty is surmounted by the addition of an acid —a practice which is harmless if a tonic only is indicated, but is highly objectionable when large doses are given in fever. Other salts of quinine, however, are quite soluble, and the following table will serve as a guide to a choice of preparation :—

Name of Salt.	Percentage of the Alkaloid in the Salt.	Solubility in Cold Water.	Equivalent Value to Quinine Sulphate (Quinine Sulphate = 1).
The alkaloid is:—		In parts.	
Quinina, $C_{20}H_{24}N_2O_2 3H_2O$.			
Sulphate, $(C_{20}H_{24}N_2O_2)_2H_2SO_4 15H_2O$	73·5	800	..
Hydrochlorate, $C_{20}H_{24}N_2O_2HCl2H_2O$. . .	81·8	40	·9
Bihydrochlorate, $C_{20}H_{24}N_2O_2 2HCl 3H_2O$. .	72·0	1	1·02
Hydrobromate, $C_{20}H_{24}N_2O_2HBrH_2O$, . .	76·0	45	·96
Bihydrobromate, $C_{20}H_{24}N_2O_2 2HBr 3H_2O$, .	60·0	7	1·23
Bisulphate, $C_{20}H_{24}N_2O_2H_2SO_4 7H_2O$, .	59·1	11	1·24
Phosphate, $(C_{20}H_{24}N_2O_2)_3(H_3PO_4)_2 6H_2O$,	72·8	78	1·01
Valerianate, $C_{20}H_{24}N_2O_2C_5H_{10}O_2$, . .	75·7	110	·97
Lactate, $C_{20}H_{24}N_2O_2C_3H_6O_3$. . .	78·2	10	·94
Salicylate, $C_{20}H_{24}N_2O_2C_7H_6O_3$, . .	70·1	225	1·05
Hydrochlorosulphate, .	74·3	2	·99
Arseniate, $C_{20}H_{24}N_2O_2AsH_3O_4H_2O$, .	69·4	Slightly soluble	1·06

The *treatment of benign tertian and quartan infections* should consist of:—

R.—Quin. bihydrochlor., ... gr. v.
Aq. chlorof. ad . . . . . . ℥i.

M. Ft. Mist.

Sig.—℥i. when the temperature is normal, and continue twice daily for a week.

If the fever is at an end by then, the following pill should be prescribed:—

R.—Ferri hypophosph., ... gr. ij.
Quin. bisulph., ... gr. i.
Acid arseniosi, ... gr. $\frac{1}{50}$.

M. Ft. pil. i.

Sig.—One pill to be taken thrice daily after food for fourteen days.

The *treatment of malignant infections* must be prompt.

Ten grains of quinine should be immediately administered, by the

mouth if possible, followed by 5 grains every four hours for a week, with gradual reduction.

If the patient is unconscious, or comatose, or has severe gastric symptoms, then the quinine should be administered intramuscularly, or by intravenous injection:—

℞.—Quin. bihydrochlor.,	. . . .	gr. xv.
Sod. chlor.,	. . . . . .	gr. i.
Aq. destil.,	. . . . . .	℥ijss.

M. Ft. Sol.

Sig.—The solution to be injected intravenously (after previous warming), and repeated, if necessary, several times at intervals of four hours; or:—

℞.—Quin bihydrochlor.,	. . . .	gr. xv.
Aq. destil.,	. . . . . .	℥i.

M. Ft. Sol. hypoderm.

Sig.—The solution to be injected deeply into the muscles of the scapular or gluteal region, and the dose repeated, if necessary, several times.

Note.—This has been said to cause abscesses, but should not do so if antiseptic precautions are taken.

Other Drugs.—Other drugs besides quinine have been advocated from time to time, but with such an invaluable specific no other drug is necessary or advisable.

The drug to which some attention has been turned is *methylene blue* (or tetramethylthionine hydrochloride). It is claimed that a certain specific action is exerted by this substance on the malarial parasite. The dose is 1 to 5 grains, but its use need scarcely be attempted.

Many *quinine esters* and other addition compounds have been put before the public. Such are:—

1. Euquinine = Q. ethylcarbonate, 5 to 10 grains. Soluble in alcohol.
2. Acetylquinine. Has a vile taste.
3. Benzoylquinine. Quite inert.
4. Phosphorylquinine. Quite inert.
5. Saloquinine, 15 to 30 grains. Soluble in alcohol.
6. Quinine camphorate, 1 to 10 grains. Soluble in alcohol.
7. Q. bihydrochlorocarbamide, 5 to 15 grains. Soluble in water.
8. Q. saccharinate. Insoluble.
9. Q. sulphocarbolate, 1 to 5 grains. Soluble in alcohol.
10. Q. sulphocresotate, 1 to 5 grains. Soluble in water.
11. Q. vanadate.

Of these euquinine is alone worthy of attention.

Another malarial remedy with a quinine basis, and which is of undoubted benefit in many cases, is the celebrated *Warburg's tincture*, of which the formula is:—

℞.—Aloes socot.,	4
Rhei rad.,	4
Angelicæ sem.,	4
Conf. damocratis,	4
Helenii rad.,	2
Croci sativi,	2
Fœniculi sem.,	2
Cret. præp.,	2
Gentian rad.,	1
Ledoariæ rad.,	1
Pip. cubebæ,	1
Myrrh elect.,	1
Camphoræ,	1
Bolet. laricis,	1

Digest c̄ 500 proof spirit on water bath for twelve hours; express; add:—

Quin. sulph.,	10

Continue heating till quinine is dissolved, and, when cold, filter.

Sig.—The Warburg's tincture.

In prescribing *quinine* it should be remembered that its *incompatibles* are:—

(*a*) Alkalies.
(*b*) Alkaline carbonate.
(*c*) Astringents.

The *treatment of malarial cachexia* should be directed towards a general restoration of the system.

The patient should be removed to a healthy locality.

Small doses of *quinine*, *arsenic*, and *iron* should be given—such as the pill mentioned previously.

Rest and fresh air should be enjoined and excessive exercise forbidden. Great care should be taken to avoid chills or over-fatigue.

The diet should be nutritious, but simple. The summer should be passed at a bracing English sea-side place. The spring and autumn are suitable times for benefit from spas, such as Kissingen or Homburg; but the winter should be spent on the Italian Riviera.

Merck's syrup is an excellent tonic in malarial cachexia:—

℞.—Quin. sulph.,	
Cinchoninæ sulph., āā,	ʒij.
Ac. sulph. dil., q.s. to dissolve,	
Ol. aurantii, q.s. to flavour,	
Saccharin,	gr. viij.
Sp. vini. rect.,	
Glycerin, āā,	*f.* ℥ij.
Syrup ad	*f.* ℥xvj.

M.—Ft. Syrup.

Sig.—℥ss. t. d. s. p. c.

For the paroxysmal headache and neuralgia so common after malaria, the following is recommended :—

℞.—Quin. hydrochlor., ...	gr. iij.
Tct. cimicifugæ, ...	♏v.
Caffein cit., ...	gr. ij.
Sp. chlorof., ...	♏x.
Inf. aurant. Co. ad	℥i.

M.—Ft. Mist.

Sig.—℥i. b. d. s.

General management of malaria.—Rest in bed; blankets for cold stage; sponging for hyperpyrexia; nutritious slop diet; and plenty of drinking water.

Prophylaxis.—In malarious neighbourhoods use a mosquito net. Do not live too near natives.

Search out and abolish any breeding grounds for mosquitoes.

Take quinine, either in 15 grain doses every eight days; or, better, in 2 grain doses every morning.

Public prophylaxis falls mainly under the following heads :—

Mosquito campaigns.
Instruction of natives.
Isolation of cases.
Facilities to natives for obtaining quinine.

CHAPTER XXIX.

MALTA FEVER.

Definition.—A specific infectious fever of extended duration and frequent relapses, characterised by copious diaphoresis and frequently accompanied by articular rheumatism and orchitis.

Synonyms.—*Mediterranean fever*, *Gibraltar fever*, *Rock fever*, *Gastric remittent* and *Bilious remittent fever*, *Intermittent typhoid*, &c.

History and Distribution.—The disease is first mentioned in the British sanitary reports at the beginning of the nineteenth century, but was not definitely recognised as a specific illness until described by *Marston* in 1859, who called it "Bilious remittent fever."

Previous to that time it had been confounded with typhoid or malaria. Subsequent researches resulted in the discovery by *Bruce* (at Malta) in 1887, of the specific organism, the *Micrococcus melitensis*.

The name given to the disease is somewhat unfortunate; for, although first recognised and extremely common on the coasts and islands of the Mediterranean Sea, it has also been described by *Buchanan*, *Lamb*, and *Pai*, as occurring in India; while the recent investigations of *Bruce*, *Wright*, and others tend to show that it also occurs in the West Indies and North and South America. *Manson* has noted cases with the same clinical symptoms in China; and has also seen two cases, giving the serum reaction, which originated in England.

Etiology.—The discovery of the *Micrococcus melitensis* by *Bruce* has been confirmed by *Hughes*, *Wright*, *Durham*, and others.

The following are the chief points with regard to this micro-organism:—

1. It is a very small round or oval micrococcus.
2. It is about $\frac{1}{3}$ μ in diameter.
3. It is often seen in couples, and sometimes in chains of four.
4. Is non-motile.
5. Is readily stained with basic dyes.
6. Is decolourised by Gram.
7. Does not ferment glucose as do other streptococci that occur in the gut.
8. Renders milk alkaline without coagulation (other streptococci curdle milk).

9. Is agglutinated by a specific serum in dilutions of 1 in 1,000.
10. It withstands desiccation for some time.
11. Meat peptone (agar $1\frac{1}{2}$ per cent.) proves a good culture medium.
12. Litmus-glucose-nutrose agar facilitates isolation of the coccus.
13. Is of slow growth, and forms rosettc-shaped white colonies, becoming tan colour after a few months.
14. Inoculations of pure cultures will give rise to the same disease in monkeys and other animals.
15. It is scarcely to be found in the peripheral circulation,
16. but occurs in large numbers in the spleen and liver.
17. It is stated to occur in the milk of most of the Maltese goats.

The **method of transmission** of the diseasc is uncertain.

That the specific coccus occurs in fresh *goat-milk* might indicate a possible channel of infection.

Most authorities are agreed that the disease is *not contagious.*

Manson considers the weight of evidence to point to its diffusion by *air currents* rather than by food and water.

The *mosquito* as an intermediate host has been weighed in the balance, and so far found wanting.

At the instigation of *Ross*, a series of interesting experiments were carried out in 1904 by two naval surgeons (*G. M. Levick* and *E. H. Ross*). Non-immunes were in close contact with, and even slept in the bed of, two severe cases, on two different ships. No cases occurred before in these ships, and none afterwards, nor did these attendants contract the disease by this direct contact of fomites, &c. The second experiment was connected with transmission by inhalation, infected dust, &c.

The urine of a patient was mixed with dust, and, when dry, both inhaled and swallowed. A living culture of the organism was then dried and powdered; after which it was both inhaled and swallowed by the same non-immune. Both the above experiments gave negative results.

The third experiment consisted of the drinking by two non-immunes of a tumblerful of a mixture of 10 ozs. of the urine of a Malta-fever patient mixed with 5 gallons of drinking water. The disease was not contracted by either.

The fourth experiment was made with mosquitoes. A *Stegomyia fasciata* and a *Stegomyia pseudotæniata* were allowed repeatedly to bite cases of Malta fever at every stage of the disease, and afterwards repeatedly to bite two non-immunes. The result was negative in all cases. Similar experiments were attempted with *Culex pipiens*, *Culex fatigans*, and the sea-water bred species *Acartomyia zammittii* and *Theobaldia spathepalpis*, but so far the attempt has failed, owing to the difficulty of making these species feed on blood when in captivity.

It has been stated that the *Acartomyia* has the same geographical distribution as Malta fever.

It will probably not be long before the exact means of transmission will be determined experimentally.

The **incubation period** as proved by intentional and accidental inoculation has proved to vary from *five to fifteen days.*

The **predisposing causes** are :—

1. Hot, dry weather.
2. Sewage contamination.
3. Overcrowding
4. Chills and excesses.
5. Youth (6 to 30 years).
6. Recent arrival in endemic area.

A considerable immunity is acquired by one attack.

Differential Diagnosis.—The ineffectiveness of quinine and the absence of parasites in the blood will exclude malaria. The absence of spirilla in the blood will negative relapsing fever.

The absence of rose-spots and diarrhœa, and the presence of articular trouble and sweats, should prevent confusion with typhoid. Liver abscess should be thought of, and phthisis should be excluded.

The only certain confirmation, however, is by serum agglutination, for which dead cultures may be used, and can, therefore, conveniently be kept in stock. In the later cachectic stages, the reaction falls low and may be lost. It is of the utmost importance that the cultures used should be perfectly reliable.

Symptoms.—After a series of prodromata, such as listlessness, anorexia, sleeplessness, headaches, &c., the symptoms become more urgent, and the onset of fever is not infrequently accompanied by severe headache, facial congestion, tinnitus aurium, and epistaxis. The tongue is coated and flabby, with red edges and tip. The tonsils and pharynx are congested. There is nausea, anorexia, and constipation. If errors in diet have been made, constipation may give way to a dark, offensive diarrhœa, with blood-stained stools.

As a rule, patients will complain of palpitation and of epigastric oppression and tenderness.

During this time the spleen and liver will be found to be enlarged, and almost invariably a slight cough with scanty expectoration is developed.

Meanwhile the temperature rises gradually, being higher in the evening than morning, until the maximum is attained in three or four days. Profuse diaphoresis occurs, usually about 1 or 2 in the morning, and a large crop of sudamina are consequently to be found. Consciousness is generally maintained, though slight delirium may occur at night.

After the first week or two, the headache and acute symptoms usually abate; the temperature gradually falls, and the patient begins to think himself convalescent: his aspect is natural, but rather listless; his tongue is cleaner, and the appetite returns. The sweating, however, continues; his complexion gets sallow or clay-

coloured; and, in a few days, the first of a long series of relapses sets in with a rise of temperature. Weakness, apathy, and emaciation become more marked, and an increasing anæmia is obvious.

The temperature may range high, but, as a rule, the patient sleeps well, and has no delirium or restlessness.

It is during this further course of the illness that rheumatoid articular affections are developed. One or more joints in sequence become red, swollen, and intensely painful, according to *Bruce*, in about half the cases.

Orchitis may occur for a day or two, and then vanish. Neuralgia may develop in any part of the body.

As pointed out by *Bassett-Smith*, in the later stages of the disease the effect of the toxins evolved by the micrococcus is that of a powerful cardiac poison somewhat analogous to that of diphtheria, and it is usual to find a fast pulse, irregular palpitation, and excitable action of the heart on slight exertion or mental perturbation.

Albuminuria is rare.

Wave of fever follows wave, lasting for many weeks, or even months, the length and intensity of each attack gradually diminishing and becoming a remittent rather than a continuous type as at first. As the temperature reaches and stays at normal the body weight and strength increase and the anæmia diminishes.

Hughes gives the average length of stay for hospital patients as 90 days.

More rarely cases may be mild and not relapse, lasting not more than two or three weeks. On the other hand, it may be so severe as to be indistinguishable from a fatal case of typhoid. *Hughes* distinguishes three types:—(1) The undulant; (2) the malignant; and (3) the intermittent, in which undulations are less marked; the duration is shorter and the complications milder.

Prognosis.—There is usually a favourable termination, the case mortality being only about 2 or 3 per cent.

A fatal termination usually occurs during the first four to six weeks, and is generally caused by hyperpyrexia.

Other complications may also prove fatal, such as pneumonia, endocarditis, or anæmia.

Pathological Anatomy.—The spleen is slightly enlarged and hyperæmic, and may be either soft or of normal consistency. There is enlargement of the Malpighian bodies.

The intestine shows a hyperæmic mucous membrane, the follicles and Peyer's patches being unchanged. Ulcers are rare, and have no connection with the illness.

The mesenteric glands are sometimes enlarged.

The liver is usually hyperæmic and the gall-bladder distended; the interlobular tissue, according to *Bruce*, shows round-celled infiltration.

The kidneys frequently exhibit glomerulo-nephritis.

The lungs are always congested, and occasionally hepatised.

Treatment.—As no specific remedy has been discovered, the treatment is purely empirical.

At the outset a dose of castor-oil or calomel is advisable.

The temperature should be kept below 103 F. by ice-pack or cold sponging or baths, and this is especially necessary if hyperpyrexia is inclined to supervene.

Throughout the febrile period only slops should be given—milk, broth, meat-juice, gruel, &c.—and solid food gradually resumed with convalescence.

Symptoms should be treated as they arise. Sulphate of quinine is comparatively valueless.

If coal-tar antipyretics are given for relief of the headache and neuralgia they should be administered with caution, as the heart is already depressed.

For the sleeplessness chloral hydrate and bromides should be tried; cardiac conditions may demand stimulants and digitalis.

The affected joints may be bandaged with hot soda compresses, and salicylate of quinine given internally.

Imperial drink and lime-juice are both of them refreshing and harmless to the patient.

The serumtherapy has, so far, not had a very extended trial. It has been tried at Netley and elsewhere with a certain number of favourable results.

When the patient enters the stage of convalescence great care should be taken to avoid a relapse. He should rest for at least three weeks after the temperature is normal.

Strengthening food, iron, or other tonics are indicated, but complete recovery seldom occurs without *change of climate*.

The voyage should not be undertaken during the height of the fever, nor straight to a winter at home.

CHAPTER XXX.

PLAGUE.

Definition.—A specific infectious disease, running an acute course, and characterised by high fever, severe constitutional disturbance, and either adenitis, pneumonia, or septicæmia.

Synonyms.—*Pestis bubonica*, *Clades inguinaria*, *Black-death*, *Malignant polyadenitis*, *La peste* (Fr.), *die Peste* (Germ.), *la plaga* (Span.), *la pestilenza* (Ital.).

History and Geographical Distribution.—The history of plague can be traced back to the second century B.C., but such history is somewhat nebulous, and any epidemic disease with a high mortality would probably be called plague. Epidemics of plague in Syria and Egypt are mentioned by *Rufus* of Ephesus (a contemporary of Trajan, 98 to 117 A.D.). The next epidemic of importance spread over the whole of Europe at the time of Justinian (A.D. 527 to 565), and lasted for some fifty odd years.

Numerous epidemics occurred in various parts of the world during the subsequent centuries.

In some Indian writings of the twelfth century plague is evidently referred to, and instructions given to leave any place when rats die in numbers.

The next most fatal visitation in Europe was the great Black-death of the fourteenth century. From Greenland to Africa, from Europe to China the great pandemic raged, and many millions succumbed.

In the second half of the seventeenth century another pandemic visited Europe, and is renowned in English history as the great plague of London (1664-65), when 70,000 of the 460,000 inhabitants perished. To this day, in City Road, can be seen the circular iron railing surrounding the green grass plot which marks one of the great burial pits, then outside the city gates.

Probably all the European epidemics were imported from the East.

Scheube thinks there is reason to regard *Southern Thibet* as an original centre.

After the seventeenth century outbreaks of plague seemed gradually to cease, and Europe was almost left alone.

The great modern pandemic apparently began in the Chinese province of Yunnan, where *Rocher* reports it in the town of Mengtsz, in 1878. Within ten years it had reached Pakhoi, and spread thence to Kwang-tung in March, 1894. Two months later Hong-kong and Canton were infected, and, a little later, it spread to Amoy. In 1895, Swatow, Foochow, and Formosa were visited.

In 1896 it appeared in Bombay. It spread to Calcutta while the author was there in 1897.

Along trade routes this Indo-China epidemic then spread, reaching Madagascar, Mauritius, Delagoa Bay, the Philippines, Japan, Australia, Suez, Alexandria, the Pacific Islands, Paraguay, Brazil, and Argentina; also limited outbreaks at Oporto and Glasgow, with one or two imported cases at London, Plymouth, Hamburg, Cardiff, Trieste, &c.

The extent of this epidemic may be realised when it is seen that there were 1,040,429 deaths from that cause in India during 1904.

Etiology.—The cause of plague is a specific micro-organism called *Bacillus pestis* (Fig. 59). This was first discovered during

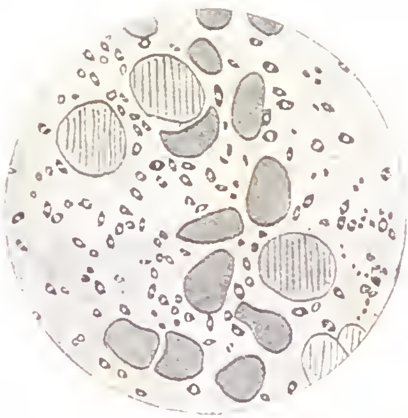


Fig. 59.—Plague bacilli from bubonic smear.

the Hongkong epidemic in 1894, when they were seen and recognised by *Kitasato*.

Very shortly afterwards, and independently, *Yersin* discovered the same bacillus, and he was probably the first to isolate and describe what was really a pure culture of the bacillus (*vide* extracts from Kitasato's Japanese publications, contained in the Formosa reports of Yamagima and Ogata; and Yersin's report to the French Academy).

The bacillus is met with in the buboes, spleen, lungs, liver, kidneys, walls of the stomach and intestines, and in other viscera.

In the peripheral blood it is only met with in septicæmic cases, or towards the end of any fatal case.

In the buboes the organism occurs as a nearly pure culture; but, as suppuration takes place, it gradually disappears.

Microscopically it is a short, round-ended rod—almost a coccoid or oval shape—measuring 1·5 μ to 2·0 μ in length by 0·7 μ in breadth (*Hewlett*).

It is usually regarded as non-motile. *Kitasato* states that, if kept at a temperature of 37° C., it is actively motile.

Gordon, *Muir*, and others have demonstrated flagella—spiral, terminal, and numbering either one, two, or three.

The bacilli generally occur singly, but may be in pairs or chains, especially in the sputum.

Involution forms are frequently met with, either thickened, pear-shaped, or spindle-shaped, especially when the bacilli exist under unfavourable conditions, such as mixed infectious, old cultures, &c. (*Rees*).

It is a *facultative anaërobe*. It has no true capsule, and *does not form spores*.

Bipolar staining is easily obtained with any ordinary stains.

The bacillus is *decolourised by Gram*.

Cultural Characteristics.—The organism grows well on most laboratory media.

On *agar* or *glycerine agar* the colonies are grey-white and discrete; afterwards coalescing and forming an abundant, moist, cream-coloured, or grey-white growth, with a crenated appearance.

In *bouillon* a preliminary turbidity gives place to the formation of a flocculent deposit on the sides and bottom of the tubes.

In *gelatin* no liquefaction occurs, and the growth is rather limited, opaque, white, and with irregular margins.

On *blood serum* it grows rapidly, and in twenty-four to forty-eight hours a slightly elevated, moist, yellow-grey, abundant growth appears.

On *potato* it grows, but not characteristically.

In *sugar-agar* it forms a small amount of acid.

In *milk* coagulation is caused in about two weeks (*Klein*).

An absolutely diagnostic cultural peculiarity was, however, discovered by *Haffkine*.

The cultures are sown upon a neutral or faintly alkaline peptone broth, and a few drops of clarified olive oil or other fat are added. The flask is kept absolutely at rest. In ten to twelve hours there is a diffuse cloudiness, which is gradually replaced by a stalactitic growth dependent from the under surface of the fat. If shaken, these will fall to the bottom like snow-flakes, and more will be formed on re-incubating.

Animal Inoculations.—Apes and rodents, such as rats, mice, guinea-pigs, &c., are highly susceptible; in one to three days there is anorexia, depression, hurried respirations, paresis of hind limbs, and usually convulsions, followed by death, bacilli being found in large numbers in the spleen, liver, and blood.

Sheep, goats, cows, and horses are only slightly susceptible; dogs and cats even less so.

Proof of the Connection between the Bacillus and the Disease.—1. It is constantly present in a person suffering from the disease.

2. If the infective material from a case is inoculated into a

susceptible animal, the animal sickens and dies, and the bacillus can be isolated and can re-convey the disease to another animal.

3. Direct inoculation conveys the disease.

In 1802, *Whyte* infected himself, and died.

In 1835, two condemned criminals in Cairo were inoculated with plague blood, contracted the disease, but recovered.

In 1894, two Japanese doctors contracted the disease in Hongkong by wounds of fingers.

In 1898, in a Vienna laboratory, *Müller* contracted the disease.

4. The bacillus can be cultivated, and the cultivations will produce a similar disease.

Effects of Disinfectants, &c.—The bacillus is extremely resistant to cold, and may be frozen for four months without being killed.

Laboratory cultures soon lose their virulence, but this may be restored by passing them through susceptible animals.

The following bactericidal figures are taken from *Hankin*, *Watkins-Pitchford*, and the German Commission:—

A thin layer exposed to direct sunlight is killed in	1 hour.
Cultures heated to 70° C.,	10 mins.
,, ,, 80° C.,	5 ,,
Treated with corrosive sublimate (1 in 5,000),	5 ,,
,, ,, potassium permanganate (1 in 5,000),	5 ,,
,, ,, chloride of lime (1 in 2,000),	5 ,,
,, ,, lysol (1 in 2,000),	5 ,,
,, ,, hydrochloric acid (1 in 1,428),	5 ,,
,, ,, quicklime (1 in 1,000),	5 ,,
,, ,, copper sulphate (1 in 1,000),	5 ,,
,, ,, izal (1 in 1,000),	5 ,,
,, ,, nitric acid (1 in 500),	5 ,,
,, ,, sulphuric acid (1 in 500),	5 ,,
,, ,, carbolic acid (1 in 50),	5 ,,

Mode of Communication.—As the organism does not possess spores, and has low powers of resistance outside the body, it is doubtful if it is ever air-borne. The following are the channels of infection :—

1. Skin or mucous membrane.
2. Respiratory tract.
3. Gastro-intestinal tract.

1. *By the Skin.*—This is by far the commonest way, and the only way in bubonic cases.

The unbroken skin, however, is not susceptible. There must be an abrasion, or loss of continuity or insect bite, &c.

The majority of cases are probably carried by fleas or other biting insects from an infected patient or an infected rat. Inoculation of a skin abrasion by plague material will also be an occasional cause, such material being either infected discharges from a patient, post-mortem handling of viscera, soiled clothes, &c. It is doubtful

if soil ever conveys the germ to an abraded foot, &c., as *Hankin* in Bombay examined numerous specimens of earth in infected houses and only found the bacillus in one doubtful case.

2. *By the Respiratory Tract.*—Under ordinary circumstances the bacillus does not exist in the air. A pneumonic case sneezing or coughing in the face of an attendant may convey the bacillus aërially; and rats may be infected by rubbing virulent cultures on the nasal mucous membrane.

3. *By the Gastro-intestinal Tract.*—In the intestines of animals fed on plague organs or infected grain, bacilli are found in large numbers. The bacillus has, moreover, been isolated from well water in the neighbourhood of an infected dwelling. Thus food and water may both convey the disease, but this channel is probably a rare one. Very likely primary septicæmic cases are thus conveyed.

When the cervical glands are affected, entrance of the bacillus has probably been *via* the tonsil.

Insects as Plague Carriers.—*Flies* were found, in 1894 by *Yersin* in Hongkong, to be infected with plague bacilli.

Ants were found by *Hankin* to have plague organisms in them, but they neither die of plague, nor retain the infection for any length of time.

La Bonadiere has demonstrated the presence of plague bacilli in a *mosquito*.

The Indian Plague Commission concluded that the case for the transfer of plague by suctorial insects from sick to healthy animals was a weak one. They pointed out, however, that the mortality amongst the Jains in Bombay was much larger than amongst other castes. These people consider all animal life as sacred. They are a wealthy people and look comparatively clean, but their tenets naturally lead to a profusion of lowly visitors, and *pediculi* have, therefore, been suggested as factors in the spread of plague.

Flies have been proved to harbour the bacillus in their intestines for some time; but experiments made in Manila, where flies were fed on plague material and subsequently attracted by syrup to shaved guinea-pigs, were in every case followed by negative results, and it is, therefore, probable that flies have no connection with the spread of plague, except perhaps a chance mechanical inoculation of a wound.

Bed-bugs may be a possible intermediary, though Nuttall considers this cause negligible.

Fleas are undoubtedly the chief means of propagation from animals to man, and man to man, in bubonic plague. Many observers have doubted this and laid much stress on the differences between human and various animal fleas, and the dislike of one species of flea for an unusual host. Most of this opposition is, I think, due to want of knowledge of the habits of rats and fleas. The subject was admirably epitomised and argued by *Liston* (of the I.M.S.) in a paper read before the Bombay Nat. Hist. Society in November, 1904.

Most varieties of fleas have different hosts, and do not, as a rule, change hosts.

We have—

Pulex irritans	of man.
P. cheopis	,, the black rat (*Mus rattus*).
P. fasciatus	,, the large brown rat (*Mus decumanus*).
P. serraticeps (or *P. felis*)	,, cats, dogs, rats, monkeys, sheep, &c.

This latter flea is somewhat peculiar, not only in affecting many hosts, but also in being of less nocturnal habit than most fleas.

The habits of fleas are such that if their host dies they leave the body when it becomes, or is nearly, cold and seek a new host, if possible of the same kind; but if not, the nearest warm-blooded creature.

The habits of rats are as follows:—They live in colonies and are very gregarious. The black rat (*Mus rattus*) is a house rat and lives with or near man. The large brown rat (*Mus decumanus*) does not so closely follow human fortunes. It also is, to a certain extent, a house rat, but lives in roofs, cellars, drains, &c., and only comes near the haunts of men when in search of food.

Both these kinds of rats are equally susceptible to plague.

When an epidemic of any kind occurs amongst them, and deaths take place in any number, they take fright and migrate *en masse* to a new neighbourhood, leaving the sick and dying behind them.

The fleas which have been feeding on these sick rats, in due course leave the dying and dead; and, having none of the same kind of host, for the sake of food must perforce seek a different host or die.

If this train of circumstances occurs in the case of *Mus rattus*, then the escaping fleas will probably find a human host as the nearest source of food. If, however, the plague has been amongst *Mus decumanus*, which lives in drains and cellars, &c., the chances that the flea will find a human host are enormously minimised.

This will largely account for vagaries in the spread of plague.

When plague ravaged Europe up to the seventeenth century the black rat was of universal distribution in that continent. About that time the brown rat was introduced, multiplied with enormous rapidity, displaced the black rat, and plague gradually disappeared.

The two small outbreaks of plague in Glasgow are examples of limited spread when the source has been a brown-rat epidemic. In the autumn of 1900, chiefly in three houses in Glasgow, there occurred 36 plague cases and 16 deaths. The origin could not be traced and no rats were to be found. A year later, a second outbreak occurred, this time in a rag store where dead brown rats were found. The only human beings attacked were five in the rag store and five in the cellars of the Central Hotel. In the basement of a house in Gordon Street, *Warren* found 67 dead brown rats, of which 40 had plague. For the next two years brown rats with

plague were found, but no more cases were seen amongst human beings. Now had the plague been epidemic amongst the black house rat, the chances are that the disease would have spread much more; but as it was confined to the brown cellar or drain rat it exactly followed the course we should have foretold.

The following observations of Liston in 1903 show the migration of the black-rat flea—*P. cheopis*:—

Some guinea-pigs died of plague and were found to be covered with *P. cheopis*. Dead black rats had previously been found lying near the cage.

In an Indian house black rats had been dying in considerable numbers and then suddenly disappeared, and almost immediately the inmates were invaded by fleas. Plague broke out amongst them, and out of 30 fleas caught on them, 14 proved to be *P. cheopis*; although previously Liston had only found one *P. cheopis* out of 246 fleas found on human beings.

It is probably to some such factors as these, rather than to any climatic influences, that the apparent immunity is due of such places as Singapore, where the black rat is scarcely seen.

Clothing or Food, &c., Plague Carriers.

Clothing.—*Wilm* has found that a sterilised piece of clothing after being soaked in a bouillon culture of the bacillus and afterwards protected, gave cultures after four weeks.

A further example of infected clothing is given by *Edwards*. In 1900, a family came from a plague-stricken district in Mauritius to Natal. Two months later one of the sons, aged 16, opened a box of soiled linen which they had brought with them, in two days plague declared itself and he was dead in 48 hours.

Clothing will carry infection in two ways:—Either the bacillus will gain access directly through a skin abrasion; or else parasites will infect themselves and in turn infect the wearer.

Grain and Foodstuffs.—All foodstuffs shipped at any infected port should be viewed with suspicion; not because in themselves they are necessarily dangerous, since intentionally infected grain is innocuous after four to six days, but rather because of their liability to harbour rats, mice, &c. Rotten grain inhibits the growth of the germ even more than does fresh grain.

Climatic and Personal Factors.—Plague may occur at any *season*, hot or cold. In Hongkong there is an annual exacerbation beginning about March or April, and ending about September (*i.e.*, during the hot season). Neither *age* nor *sex* appear to have anything to do with the incidence, though perhaps it is less common in the aged.

Nor has *race* any influence. According to *Cantlie* the Malay race are wonderfully exempt; but when it is considered that Malay trade with infected areas is small: that there is no single populous Malay town in the Archipelago or Peninsula analogous to any Indian or

Chinese city; that the black rat is rare, and that the introduction of infection is infrequent, then we can hardly consider that the susceptibility of the race has been put to a fair trial.

Method of obtaining Material.—*Bubonic Cases.*—Cleanse the bubo and insert a hypodermic needle; spread the blood or exudate so obtained on a slide; dry in air; fix by passing through flame three or four times. Stain with carbol fuchsin.

Pneumonic Cases.—Smear some sputum on a slide thinly; dry and proceed as above.

Septicæmic Cases.—Prepare a blood film, sec. art.

Symptoms.—Various *types of disease* have been recorded, such as bubonic, pneumonic, septicæmic, intestinal, nervous, puerperal, typhus, Pestis fulminans, Pestis ambulans, and Pestis minor.

For practical purposes, however, there are only four types:—

1. Pestis minor.
2. Bubonic plague.
3. Pneumonic plague.
4. Septicæmic plague.

Incubation Period.—This is a matter of some importance. Any time from two to fifteen days has been put forward. As in all other such diseases there are innumerable sources of error. The author has seen a case occur in a passenger on a coolie ship on the twelfth day after leaving an infected port. This does not necessarily mean that the incubation period is eleven days; the probability in such case is that some infected material happened to have inoculated the patient a few days before the onset of symptoms.

The only sure guide on the subject is the observed result of accidental or experimental innoculation. This has proved to be *one to three days.*

But not on this account should we insist on a maximum of five days' quarantine as do the modern sanitary conventions. When we are dealing with bodies of men, such as coolie immigrants, pilgrims, troops, native deck passengers, &c., whose persons and effects have not been *thoroughly* disinfected before leaving the infected area, we have no guarantee that they are not liable to catch the disease from infected material at any time subsequent to their departure. Every day beyond five days will, of course, lessen the probability; therefore, in all these cases it is as well to insist on ten days' quarantine from an infected port, and *also* thorough disinfection of persons and effects before allowing them to land.

Invasion.—Plague generally exhibits itself very suddenly in one of three ways:—

1. A rigor, with headache, giddiness, high fever, &c.
2. Loss of appetite, headache, fever, &c.
3. Convulsions (in children).

Prodromata, such as malaise, depression, nausea, diarrhœa, may be present, but are not common.

General Clinical Features.—Once seen, the appearance of a typical plague case is hard to be forgotten. We have—

High temperature.
Accelerated respiration.
Dry and hot skin.
Flushed face, and injected conjunctivæ.
Very anxious expression.
If able to walk there is a reeling drunken gait.
The tongue is swollen and furred with a red tip and edges.
The pulse is fast, very full, but of unexpectedly low tension.
A gland (most often in the groin) will be found swollen, hot, painful, and indurated.
If able to speak, intense frontal headache is complained of.

Such is a usual clinical picture of a bubonic case. Constipation is the rule, though diarrhœa is occasionally seen.

The temperature rises rapidly, reaching its maximum on the second or third day.

The expression of anxiety gives way to one of apathy, due to loss of facial nerve control. The speech becomes thicker. The eyelids fail to close. The pulse becomes dichrotic. The urine is scanty and high-coloured. The flushed face becomes of dusky hue.

In severe and rapidly fatal cases there is often a crisis on the day of, or the day after, the maximum temperature, resulting in a subnormal fall, collapse, and death.

Bubonic cases are not usually so rapidly fatal as are pneumonic.

If the first fall of temperature is not below 100°, and tends to rise again immediately, the probability is that the organism is invading the blood stream, and the chart will show the usual septicæmic features, with a practically hopeless prognosis.

In favourable cases there may, or may not, be a secondary exacerbation of the fever, but the course of the temperature will tend towards the normal by lysis lasting for seven or eight days.

The *cause of death* in fatal cases is generally complicated, and due to one or more of the following factors:—

Exhaustion from hæmorrhages.
Syncope from sudden exertion.
Hyperpyrexial asthenia.
Pulmonary involvement.
Bacterial toxæmia.

Whatever the type of plague, bacilli will be found in the peripheral blood just before death.

The Pneumonic Type.—Attention was first called to this type by *Childe*, who found that deaths from pneumonia increased as plague increased.

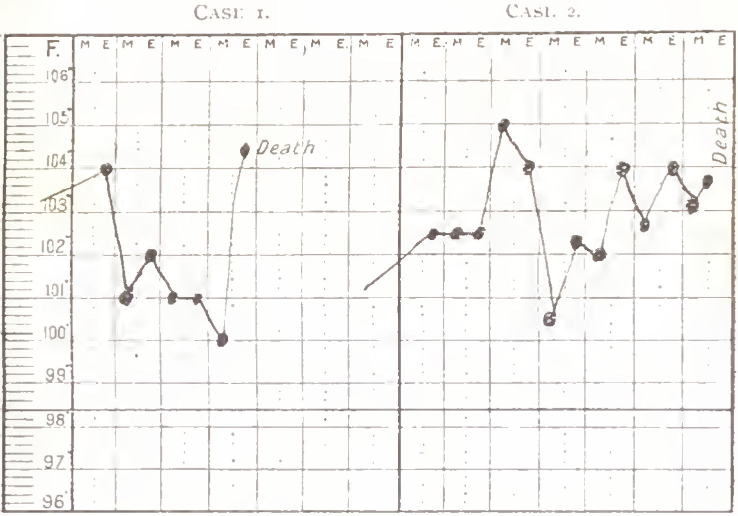
The attack in a primary case is ushered in by a slight rigor, dizziness, headache, and a rise of temperature (Fig. 60).

Respiration becomes accelerated, and a tightness is felt in the chest, accompanied by a cough. Dulness and rhonchi can be made out. Fever runs high, with a low-tension pulse. Prostration is extreme. No herpes is present. Cyanosis is frequently observed.

The sputum is not rusty coloured as in ordinary pneumonia, but may be sanguineous, and always displays a nearly pure culture of plague bacilli.

The disease is very fatal, and naturally very infectious.

Occasionally a secondary pneumonia occurs in the course of an ordinary bubonic case.



Pneumonic plague of right lung—no bacilli in blood during life.

Bubonic plague. Secondary septicæmia. Bacilli in blood before death.

Fig. 60.

Pestis Minor.—This is a mild form of the disease, characterised by slight fever, enlargement of one or more glands, and practically no constitutional disturbance.

Simpson observed that it frequently preceded an epidemic. It may also follow an outbreak, as an exemplification of diminished virulence.

From the mildness of the symptoms it is obvious that many cases will pass unrecognised, and be a grave source of danger.

The author had a striking example of this in an epidemic which occurred in 1903 amongst some 900 coolies landed from an infected ship on the Quarantine Station at Singapore.

Ten deaths had occurred during the voyage from Amoy, and the cases which supervened during their quarantine will be seen from the accompanying table.

It is noteworthy that Pestis minor did not appear until the virulence of the small epidemic was declining.

Date.	Bubon.	Pneum.	Septic.	Minor.
July 6, . .	2	...	1	...
,, 7, . .	...	...	...	...
,, 8, . .	...	...	1	...
,, 9, . .	1	3	1	...
,, 10, . .	2	1	3	...
,, 11, . .	2	...	...	34
,, 12, . .	...	...	...	30
,, 13, . .	...	...	...	1
,, 14, . .	...	1	2	28
,, 15, . .	1	...	...	3
,, 16, . .	...	...	...	6
,, 22, . .	1	...	...	...
Total, . .	9	5	8	102

This might either be a manifestation of the disease in those who were partially immune, or else a lessened bacillary virulence, such as generally occurs towards the end of a cholera epidemic.

Smears were taken from many of the buboes in the above Pestis minor cases, and in about one-third of them *Bacillus pestis* was found. In the ordinary course these cases would have passed entirely unrecognised; but daily examination of the contacts disclosed a sudden slight rise of temperature, accompanied by a tender but small enlargement of a gland.

These cases arc just as dangerous as the more severe cases, from an epidemiological point of view, and it behoves us, therefore, to be very much on the look out for them.

Some depression and cardiac excitability may remain, with or without physical weakness.

Unless (as is but rarely the case) the graver type of the disease supervenes, the mortality is practically *nil* (Fig. 61).

The accompanying chart will show the usual course of a Pestis minor pyrexia.

Complications and Sequelæ.—In almost all cases convalescence is protracted by weeks of mental and physical prostration.

Chronic sloughing of the bubo, often laying bare the deep structures or bone, is by no means uncommon.

Aphasia may follow recovery; and not infrequently petechiæ or boils are found.

Leumann mentions two cases each of paraplegia, hemiplegia, and catalepsy; also thirteen cases complicated by pregnancy, all of whom aborted, all the infants died, and only four of the mothers recovered.

Placental infection was also noted by him. A pregnant woman was attacked almost at full time. The child was born well, and the mother died of post partum hæmorrhage. Ten hours after birth buboes appeared in the child's groins and axillæ, and it died in twenty-eight hours.

Of sixty-one cases amongst nursing women, only twenty-four infants contracted plague.

Diagnosis and Prognosis.—In a well-defined case of plague the diagnosis should present no difficulty. The bubo, temperature, facies, pulse, tongue, &c., form a picture unlike any other disease.

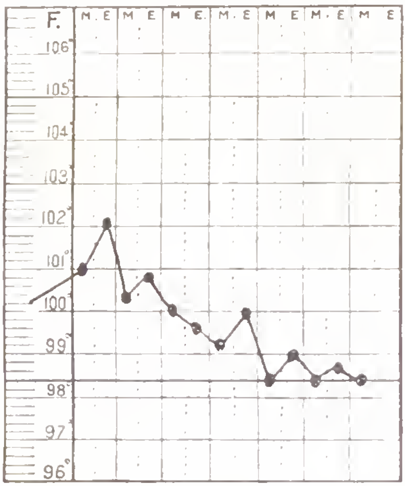


Fig. 61.—Pestis minor. Quarantine Station, Singapore. (Chinese male, aged 28.) Bubo absorbed ten days after onset. Bacilli found.

In pneumonic cases the diagnosis may be more difficult, but a microscopical examination will quickly clear it up.

The mortality is high.

In the Hongkong 1894 epidemic, the following was the plague case mortality:—

Chinese, ...	93·4 per cent.
Europeans, ...	18·2 ,,
Japanese, ...	60·0 ,,
Indians, ...	7·7 ,,

The prognosis should, therefore, always be guarded. Multiple buboes, persistent vomiting, early diarrhœa, grave cerebral symptoms, and the invasion of the blood stream by bacilli are all practically death warrants.

The first six days are the most critical time, and 70 per cent. of patients succumb during that period.

Pathological Anatomy.—Rigor mortis is usually pronounced. Post-mortem rise of temperature is not uncommon.

The most characteristic changes are met with in the lymphatic system, and a very general engorgement and hæmorrhage throughout all organs will be found.

The primary bubo shows periglandular infiltration and hæmorrhage. This œdematous infiltration may extend to the adjacent tissues. On section they appear dark red, the gland tissue being often soft and pulpy.

Microscopically, there is acute inflammation with hæmorrhages. The tissue is infiltrated with red corpuscles, leucocytes, and bacilli. The vein-walls are thinned.

The process begins in the periglandular tissue and the subcapsular lymph sinuses (*Yamagiwa*).

After the primary infection of a gland, the other lymphatics in the same chain usually become implicated.

For instance, if a femoral gland is involved the infection will spread from gland to gland up to the retroperitoneal lymphatics, and a chain of softened, swollen and hæmorrhagic glands from below Poupart up to the kidneys.

Likewise, if an axillary gland is first infected, it will spread to those of the thoracic cavity; or from cervical to mediastinal glands.

These secondary affections, however, are not so pronounced as the primary bubo.

Spleen.—This organ is infected in every case of plague. The pulp spaces prove a favourable ground for multiplication.

The whole viscus is enlarged, hyperæmic and soft.

The *lungs*, in bubonic plague, show congestion and œdema, and on section exude much blood and serous fluid. In pneumonic plague there is a typical confluent lobular pneumonia. There are areas of blackish-red hæmorrhagic patches unevenly distributed over both lungs, especially the lower lobes, exuding a sticky juice on section. Under the microscope the alveoli in the affected areas are found to be filled with blood and bacilli.

The *kidneys* are enlarged and congested with thickened cortex, and there are subcapsular hæmorrhages.

Bacilli are found in the renal tissue only in moderate numbers, but not often in such situations that their appearance in the urine is likely.

The German Commission found bacilli in the urine in only two cases.

The Austrian Commission frequently found bacilli in the kidneys, but only in five out of seventeen post-mortem urines.

The *stomach and intestines* generally show petechiæ and ecchymoses. The gastric mucosa is particularly hæmorrhagic. Microscopically the mucosa shows dilated interglandular vessels and hæmorrhages into the mucosa. Bacilli were found in these areas only in septicæmic cases (*Herzog*).

The appearance of bacilli in the fæces is even more rare than

in the urine. The German Commission were never able to demonstrate them either by culture or inoculation. The Indian Commission had the same result, but pointed out the difficulty of isolation amongst the host of intestinal organisms.

The *liver* is slightly enlarged, and very hyperæmic. There is parenchymatous degeneration, and in septicæmic cases we find greyish-white necrotic foci. Microscopically, the capillaries will be found dilated and engorged, and a granular degeneration of the parenchyma cells. Necrotic foci will show numbers of bacilli.

The following is the Indian Plague Commission's Summary of pathological conditions:—"The distinctive pathological changes produced by the virus of plague would appear to consist of universal dilatation and engorgement of veins and smaller blood-vessels, with hæmorrhages, both minute and of large amount, in nearly every part of the body; and of enlargement of the lymphatic glands, with œdema and hæmorrhage in the surrounding tissues, generally mainly implicating the external glands, but occurring likewise throughout the body and involving in a series of cases the entire system of lymphatic glands. In the lymphatic glands, the characteristic conditions are largely explainable by vascular changes, and even in the pneumonia of plague, vascular dilatations and hæmorrhagic extravasations give a special character to the lung inflammation. In no other infective disease are these features represented, but it is of some interest to note that the vascular changes, and especially the prevailing and characteristic tendency to extravasation of blood in almost every part of the body, are closely reproduced in toxæmia caused by the organic poison secreted by the venom glands of several species of serpents, such as the black snake (*Pseudechis porphyriacus*) of Australia."

Treatment—*Medicinal.*—There is no specific drug for plague. The treatment which has met with the best routine results is that of *Thomson* and *Atkinson* in Hongkong. Carbolic acid is given in large doses.

R.—Ac. carbol. pur, ...	gr. xii.
Syr. aurantii, ...	℥i.
Aq. chlorof. ad	℥i.

M. F. Mist.

Sig.—℥i. every two hours during the day.

One patient consumed over 2,500 grs. of carbolic before his blood was free from bacilli. Carbolic poisoning appears to be practically unknown. In a few cases carboluria developed, but the omission of a few doses was generally sufficient to remedy it (*Simpson*).

In 143 cases *Thomson* found that the mortality was reduced from 85·7 to 36·4 per cent. by this means.

Cyllin might perhaps be tried with advantage.

The remaining medicinal treatment is merely directed against the various symptoms as they arise.

For *hyperpyrexia*, the wet pack or sponge.

For *insomnia*, bromides.

For *cardiac debility*, hypodermics of strophanthus and strychnine should be employed (*Fraser*).

During *convalescence*, nerve tonics and hæmatinics are indicated.

Dietetic.—Solid food should never be given during the acute stage.

Local.—Do not interfere surgically with the bubo before it has suppurated.

Hot fomentations or ice-bags will afford relief.

Serumtherapy.—There are two chief curative sera—Yersin's and Lustig's; those of Terni, Bondi, and Kitasato not being much in use.

1. *Yersin's Serum.*—In 1895 at the Pasteur Institute, *Yersin*, *Calmette*, and *Borrel* immunised a horse by repeated intravenous injection of living plague cultures. The horse's serum was found to be preventive and curative for laboratory animals after six weeks.

From this horse, after a year's treatment, 26 cases were treated in Canton and Amoy. The mortality was *nil* amongst those thus treated during the first four days of illness. If no treatment was given until the fifth day, the mortality was 50 per cent.

2. *Lustig's Serum.*—The microbe is cultivated on agar plates; scraped off and dissolved in 1 per cent. sterile caustic potash. The solution is then rendered slightly acid with hydrochloric or acetic acid, and the resulting nucleo-proteid precipitate is collected on filter paper, washed and dried in vacuo. This substance is easily soluble in a weak solution of sodium carbonate. The serum from a horse which has been repeatedly inoculated for three or four months has a certain curative value. It has been tried in over 1,500 cases in Bombay, and (if septicæmic cases are excluded) the mortality is slightly reduced.

Prophylaxis. — *Haffkine's prophylactic fluid* is the one most generally employed.

The bacillus is cultivated in broth. Its purity is tested after it has done growing. It is then sterilised by heating at 65° C. for one hour and 0·5 per cent. of carbolic acld is added; after which it is drawn off into small bottles by means of a syphon.

The dose is usually from 2 to 5 c.c. (the exact amount being given on each bottle). In a few hours there is a rise of temperature to about 101° or 102° F. with a little headache, nausea, and malaise, which may continue for about 48 hours.

The accompanying chart (Fig. 62) is from observations on a certain number of nearly 2,000 injections made by the author in Singapore.

The protection lasts for some six to nine months.

The conclusions arrived at by the Plague Commission in India, on this subject, were:—

1. "That inoculation is harmless."

2. "That when given in the incubation stage—*i.e.*, before the signs of plague are apparent—it has in many cases the power of aborting the disease."

3. "That inoculation affords to all those inoculated a strong protection against attack by plague."

4. "That in the few cases when inoculated people are attacked a very large proportion recover."

Haffkine's prophylactic, if stored in a cool place, will keep for several years. The following instructions for its use are issued by the Bombay Research Laboratory:—

Instructions for the Use of the Plague Prophylactic.

"Parcel to be opened and unpacked carefully, and the corrugated paper or other packing material preserved for re-packing the empty bottles.

"Before unstoppering a bottle it must be ascertained that the rubber cork fits so tightly that it can be removed only by means of forceps.

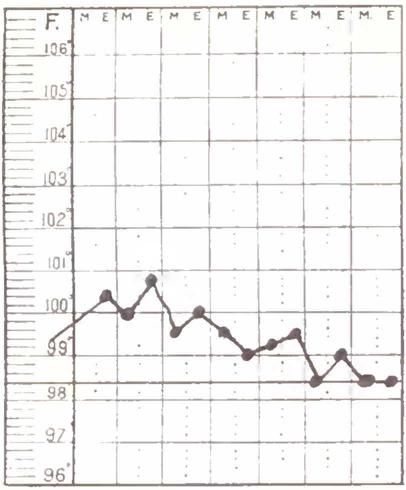


Fig. 62.—Typical inoculation reaction after injection with Haffkine's Prophylactic Serum (small dose). No. 429/03, Quarantine Station, Singapore. Chinese male. Aged 27.

"Any bottle found with the stopper not sufficiently tight, or being open, cracked, or otherwise damaged, is to be sent back to the laboratory without being used.

"Prolonged exposure of the prophylactic to daylight or to the heat of the open air is to be avoided. It should be kept packed in the box, and stored away in the darkest and coolest place available.

"The prophylactic is to be injected under the skin by means of a clean and carefully disinfected hypodermic syringe of suitable size.

"A bottle before being opened is to be thoroughly shaken so as to wash off the whole of the deposit which forms on and sticks to the bottle.

"The deposit is an essential element, and should be uniformly distributed.

"If the dose marked on the bottle is 5 c.c., then give:—

To individuals of from		
10 days to 1 year,	1/5th c.c., or	1/25th of full dose.
1 to 2 years,	1 ,,	1/5th ,,
2 to 5 years,	2 ,,	2/5ths ,,
6 to 11 years,	3 ,,	3/5ths ,,
12 to 15 years,	4 ,,	4/5ths ,,
16 to 50 years,	5 ,,	full dose.

"If the dose marked on the bottle is other than 5 c.c., the amount to be given to various ages is to be calculated in the same proportion as above.

"Women of all ages over twelve years should get $\frac{1}{10}$ less than men of corresponding ages.

"Pregnant women may be inoculated up to the seventh month inclusive without making any special reduction of dose in their case. After the seventh month, the dose should be given in two instalments separated by an interval of a week or so. Miscarriage has never been known to result from inoculation; and the danger from plague to lying-in women is so great that a special effort should be made to induce pregnant women to be inoculated.

"Persons over fifty years of age should get $\frac{1}{10}$ less for each decade above that age.

"Persons suffering from fever should not be inoculated before at least forty-eight hours have elapsed after the fever has entirely left them.

"Children stand the treatment well, and no fear need be felt in giving the doses above mentioned.

"In the case of troops and followers, and other large-bodied people, it is advisable to increase the dose by half the amount indicated on label, and to inject $7\frac{1}{2}$ c.c. if the bottle label shows a 5 c.c. dose. For strong women and children the dose may also be correspondingly increased.

"No changes in diet or occupation are necessary for the inoculated, beyond, if possible, taking some rest. Bathing in the open air should be avoided for some days. A simple purgative may be given before or within twenty-four hours after inoculation.

"It is desirable that all operations should be carefully recorded, and their effect upon the immunity of individuals, as well as upon the course of the whole epidemic watched.

"The prophylactic material is harmless, and can be thrown about without danger; but it is liable to get infected, notwithstanding the small proportion of antiseptic which it contains. A bottle, once opened, is, therefore, to be used up within an hour or so, or the rest of the contents thrown away."

CHAPTER XXXI.

SKIN DISEASES OF THE TROPICS.

Pemphigus contagiosus—Madura foot—Pityriasis nigra—Pityriasis versicolor—Tinea imbricata—Pinta—Dhobie itch—Rhezoglyphus parasiticus—Prickly heat—Cheiro-pompholyx—Pellagra—Acrodynia—Keloid—Vitiligo.

SOME of the skin diseases met with in the tropics are identical with those seen at home, such as scalp ring-worms, scabies, favus, &c.

Others have formed the subject of special chapters in this volume, such as leprosy, yaws, granuloma venerea, granuloma endemica, guinea-worm, ankylostomiasis, and ainhum.

It is proposed, therefore, in this chapter to give a preliminary tabular classification of the chief tropical skin diseases under three headings:—

1. Of vegetable origin.
2. Of animal origin.
3. Of unknown origin.

This will be followed by a more detailed consideration of those conditions which are neither well known in temperate climates, nor dealt with elsewhere herein.

Of the former class, scalp ring-worms are extremely uncommon in the tropics.

Scabies and body ring-worms, on the other hand, are enormously widespread amongst the lower native classes.

The author has examined in five years about $1\frac{1}{4}$ millions of the coolie class The very large majority of these have been of the Chinese nationality, and have been examined when entirely stripped.

Out of this enormous number only 35 cases of favus have been detected, and only one case of *Trichophyton endothrix* scalp ring-worm. No single case of *Microsporon audouïni* infection has been met with as far as is known.

Scabies was approximately present in 3·25 per cent. of the male Chinese coolies, and the lesions in a large number of the cases were so widespread that scarcely any of the body surface was unaffected.

Body ring-worms (*Tinea circinata*) due to the *Trichophyton ectothrix* were seen in some 0·65 per cent.

With regard to "dhobie itch," *Castellani* is of opinion that the trichophytic origin is different from that of *Tinea circinata*. In the subjoined schematic table, therefore, a doubtful origin has been

inserted as *Trichophyton castellanii.* The author, however, who has observed the inception of many cases, and has suffered himself from the complaint, is rather of the opinion that the *Trichophyton ectothrix* is the specific cause of both conditions. Both commence with a single small spot or patch, and spread by a circular or irregularly ring-shaped edge. The macroscopic appearance of both is identical until continued irritation and scratching, due to the situation of the dhobie itch, lead to an eczematous condition in this latter disease. In all cases, moreover, which the author has examined microscopically, the hyphomycetes were apparently identical, though no cultural differentiation was attempted.

Incidentally it may be of interest to mention that, in the above series of examinations, *psoriasis* was detected in about 650 cases; supernumerary mammæ (in males) in over 200 instances.

No case of pemphigus was observed.

TABLE OF SKIN DISEASES.

(* Dealt with in subsequent section.)

I.—Of Vegetable Origin.

Order :—**Schizomycetes.**

Family :—**Coccaceæ.**

Genus :—*Micrococcus.*

? M. pyogenes aureus, .	**Pemphigus contagiosus.*

Family :—**Bacteriaceæ.**

Genus :—*Bacillus.*

Le Dantec's bacillus, .	*Tropical phagedæna.*
B. lepræ, . . .	*Leprosy* (see chapter xxvii.)

Family :—**Cladotricheæ.**

Genus :—*Cladothrix*

C. actinomycii, . . .	*Actinomycosis.*
C. mycetomæ, . . .	*Madura-foot.*

Order :—**Blastomycetes.**

Family :—**Saccharomycetes.**

Genus :—*Saccharomyces.*

? S. litogenes. . . .	*Dermatitis.*

Order :—**Hyphomycetes.**

Genus :—*Aspergillus.*

A. fumigatus, . . .	*Epiphytic mycosis of nose and ear.*

Genus :—*Microsporon.*

M. audouini, . . .	*Ring-worm* (very rare in tropics).
M. mansoni, . . .	* *Pityriasis nigra.*
M. furfur, ...	* *Pityriasis versicolor.*

Genus :—*Trichophyton.*

T. endothrix, . . .	*Ring-worm* (rare in tropics).
T. ectothrix, . . .	*Tinea circinata.*
T. mansoni, . . .	* *Tinea imbricata.*
T. pictor, ...	* *Pinta.*
? T. castellanii, . . .	* *Dhobie itch.*

Genus :—*Achorion.*

A. Schonleini, . . .	*Favus.*

II.—Of Animal Origin.

Protozoa :—

Spirochæta pallidula, .	*Yaws* (see Chap. xxxvii.)
The Spirochete of Wise, .	*Granuloma venerea* (see Chap. xxiv.)
Leishmania donovani, .	*Granuloma endemica* (see Chap. xxiii.)

Nematoda :—

Filaria medinensis, . .	*Guinea-worm* (see Chap. xx.)
Ankylostomum duodenale, .	*Ground itch* (see Chap. xii.)

Acarina :—

Acarus scabei, . . .	*The itch.*
Rhizoglyphus parasiticus, .	* *Ulcers.*

Aphaniptera :—

Sarcopsylla penetrans, .	*Jigger* (see Chap. x.)

III.—Of Unknown Origin.

* *Prickly heat.*
* *Cheiro-pompholyx.*
* *Pellagra.*
* *Acrodynia.*
* *Keloid.*
Ainhum (see Chap. xxxvi.)
* *Vitiligo.*

PEMPHIGUS CONTAGIOSUS (*Manson*).—This skin disease is said to be fairly common in some parts of India, the Malay Peninsula, and China.

Symptoms.—The condition commences with a hemispherical tense vesicle. In a short time the fluid contents become cloudy, and the vesicle becomes flattened and flaccid.

By auto-inoculation fresh bullæ occur whenever transferred by hands or clothes.

Each bleb tends to stop spontaneously, the skin being rubbed off, and leaving a pink patch of new epidermis.

The disease is fairly common in children.

There is considerable irritation leading to scratching, and often the subsequent development of boils. The condition is apparently allied to the *Pemphigus neonatorum* of temperate climates; and, like that disease, is, in all probability, due to one of the pyogenic streptococci. Leishman Donovan bodies are reported to have been found in the fluid contents of the blebs.

The **treatment** should be effected by an antiseptic lotion applied thrice daily, and followed by an application of powdered boracic acid.

MADURA FOOT (*Mycetoma*)—**Definition.**—A disease of warm climates occurring especially in India.

It usually attacks the foot, and occasionally the hand. It rarely attacks other parts of the body, and never affects the internal organs.

It is characterised by chronic enlargement, the formation of sinuses and inflammatory degeneration.

History and Geographical Distribution.—*Kämpfer* first reported the condition in 1712. During the nineteenth century, *Godfrey*, *Balingall*, *Eyre*, and *Carter* have all studied and written about the disease. The last-named was the first to recognize the condition as a mycosis.

The chief Indian endemic districts are:—Madura, Hirsar, Ajmeer, Delhi, and other places in Kashmir, Rajputana, and the Punjab.

Cases have been reported from Cochin China, North Africa, S. America, Madagascar, Cuba, &c.

Symptoms.—An inoculation takes place through a slight abrasion. Induration results, and runs a chronic course, with or without pain. After eight or nine weeks this breaks down, forming an oozing fistula, in the discharge from which are to be seen white, red, or black granules of various sizes.

Further lesions of the same sort occur in different parts of the foot, and follow the same course until the whole foot becomes swollen, deformed, and honeycombed with sinuses.

There is seldom much pain associated with the condition.

Muscular atrophy takes place in the leg above, and the femoral glands may become enlarged from the septic absorption.

The disease may continue for ten to twenty years.

Death is usually due to exhaustion or to inter-current disease.

Morbid Anatomy and Etiology.—The condition is chiefly found in native men of middle age.

The specific cause is a schizomycete of the family Cladotricheæ—the *Cladothrix mycetomæ*. This has not yet been isolated from the soil or vegetable matter.

The fungus forms the white "fish-roe" granules most usually found in the disease. *Kanthack* considers the black variety (which is more rarely met with) to be a degeneration form of the white.

On section, the whole foot-tissues are found to be transformed into a homogeneous mass, muscles and bones being all disintegrated.

Numerous cysts and sinuses are to be seen, containing cheesy masses of the characteristic granules.

Treatment.—Iodide of potassium has no influence.

If seen early, the initial lesion should be excised; otherwise the only treatment is the surgical one of amputation, which is usually successful, as the disease is a purely local one.

Calcium iodide in 2-grain doses, three times a day, might be tried.

Diagnosis.—This should not be difficult when the disease is at all advanced.

Actinomycosis might be suspected, but the conditions are quite different (*vide* Chap. vii.).

PITYRIASIS NIGRA.—Found especially in Ceylon, the Malay Peninsula and Archipelago, Southern China, and possibly other regions.

The condition is due to a microsporon infection with the *M. mansoni* (Castellani).

This fungus has short mycelial threads 18 to 20 μ, not branching. They are about $2\frac{1}{2}$ μ broad. They are irregular, bent, or banana-shaped in outline.

The spores are globular, and mostly very large—5 to $7\frac{1}{2}$ μ, and are frequently arranged in clusters (*Castellani*).

Clinically will be found round, desquamating, dull-black patches of various sizes.

The neck is usually the favourite locality, but the disease may be found on any other part of the body, with the exception of the face, which seems exempt.

There is but little itching.

The best **treatment** is by ichthyol and sulphur.

PITYRIASIS VERSICOLOR.—On a coloured skin, this disease has usually a silver-gray appearance, though sometimes it is yellowish.

The *M. furfur* which causes the condition has considerably smaller spores than the *M. mansoni*. These spores sometimes form clusters, and sometimes do not.

The mycelium is short, thin, and regular in outline, though occasionally showing thickening or constriction.

The disease is fairly common amongst natives, and readily yields to a course of ichthyol and sulphur.

TINEA IMBRICATA (Fig. 63)—**Definition.**—A condition of the skin occurring in Eastern countries, characterised by a wide-

spread distribution of tissue-paper-like scales, and caused by a hyphomycete—the *Trichophyton mansoni*.

Synonyms.—*Tokelau ring-worm*; *Herpes desquamatus* (Turner); *Herpes farinosus* (Ritter); *Cascado* (Moluccas); *Pita* (Bowditch Island); *Gune* (Gilbert Islands); *Gogo* (Marshall Islands).

Geographical Distribution.—The disease is said to prevail in the Malay Peninsula and Archipelago and in the Pacific Islands. The author has seen many cases amongst new-coming Chinese immigrants to Singapore, chiefly from Amoy and Swatow, and to a less extent from Hongkong.

Etiology.—Manson was the first to call attention to the condition as a specific entity, in 1879.

The disease is a tricophyton infection, caused by *T. mansoni*. This fungus lies between the epidermis and rete, and grows with enormous profusion, causing the epidermis to peel up.

Microscopically it somewhat resembles *T. ectothrix*, but the mycelium is longer and much more abundant and the spores are oval.

Nieuwenhuis has cultivated it artificially on a peptone-salt-glucose-broth. *Manson* has experimentally transmitted it to healthy individuals, taking an incubation period of about nine days.

A warm damp climate is essential for its spread and growth.

All races, ages, and sexes are equally liable to infection, but naturally, on account of their habits, natives form the chief sufferers.

The transmission is either direct, or by means of clothes, mats, &c.

Symptoms.—About nine or ten days after inoculation, a slightly raised, brownish patch appears.

The centre breaks down, while the periphery extends by a spreading edge. At the site of the first brown spot another spot appears and spreads; and in this way a series of more or less concentric rings are formed; while other foci are started with similar results until quite a large area of the body may be covered with these scales.

The disease avoids the hair, crutch, axillæ, and usually the nails.

After the scales are detached, a leucodermic piebald area is left.

There is usually considerable pruritus; and the scratching which follows will often lead to impetigo or eczema.

Treatment.—Should be commenced by a hot bath and good scrub with tar soap.

Manson recommends applications of strong Lin. iodi, but usually a chrysarobin ointment (*vide* formulæ at the end of this chapter) will be found highly efficacious.

PINTA.—Definition.—A disease of warm climates, caused by a hyphomycete—the *Trichophyton pictor* (Blanchard)—characterised by piebald discolorations of the skin, without disturbance to the general health.

[*To face p.* 312.

[*Photo by Aitken.*

Fig. [illegible].—Tinea imbricata. Chinese coolie from Swatow (six months' growth).

Synonyms.—*Mal del pinto, Tinna, Quirica, spotted disease of Central America, Pannus carateus* (Alibert).

History and Geographical Distribution.—The disease has been known in Mexico since 1775.

Alibert was the first to write on the subject in 1829, and more recently *Montoya* in 1896.

It is endemic in Columbia, Venezuela, Peru, Chili, Bolivia, British Honduras, and Mexico.

Isolated cases have been reported from the Gold Coast (1901), Tripoli (1897), and by *Madden* and *Goodman* in Egypt (1899).

In Columbia it is estimated that there are usually some 200,000 cases (*i.e.*, about 4 per cent. of the population).

Etiology.—The fungus which causes the disease is the *Trichophyton pictor*, and its growth seems most favoured by hot, damp climates.

Both sexes and all ages are liable to infection, but it is commonest amongst young and middle-aged men. It is chiefly found, moreover, amongst the poorer classes, as dirt and poverty are naturally predisposing causes.

It is apparently not directly contagious, judging from the way in which it spreads amongst soldiers or families.

Montoya obtained some of the moulds from mosquitoes, and it is possible that the disease may be spread by this means. It has also been suggested that fleas or bugs may introduce them.

Successful inoculations have been carried out with rabbits, but other animals have not yet been tried.

The fungus itself consists of tapering, branched mycelial threads, to which the conidia are attached. The spores are roundish or oval, and from 8 to 12 μ in diameter.

Barbe gives nine varieties of the fungus, which account for the different colours of the eruption. The blue, black, and yellow forms are only anomalies. There are three differential forms :—

1. *Violet.*—Affects the epidermis. Seen in negroes and mulattoes of rural and mining districts. Begins on face or arms or legs. May attack mucous membranes. Loss of pigmentation late.

2. *Red.*—Affects the rete mucosum and corium. Seen in towns amongst white people. Skin smarts, and is dry. No secondary loss of pigment.

3. *White.*—Affects the rete mucosum and corium. Seen in towns amongst half-castes.

Symptoms.—There is an indefinite *incubation period* of about a month.

A slight itching precedes the eruption, which usually commences on the face or neck.

The rest of the body is affected later, with the exception of the palmar and plantar surfaces, which escape.

The eruption begins with asymmetrical spots or patches, accompanied by a furfuraceous desquamation. These spots increase in size, and gradually coalesce with others to form extensive patches, which show the characteristic colours in two or three years.

One or more colours may be seen on the same patient.

The white variety is often confused with leucodermia (vitiligo), which disease is seen both in the East and West; the latter is sometimes hereditary; is often symmetrical, with convex borders, and is not dependent on a fungus.

During this chromatic stage, the mucous membranes of mouth, prepuce, and vagina may become coloured. At a later stage the pigment disappears gradually from the centre to the periphery, the first sites to become leucodermic being such exterior surfaces as are specially subject to friction.

Course.—There are no constitutional symptoms, and the death rate is *nil*. The disease, if untreated, may last for very many years. If no spores are found in long-standing cases, the parasite is probably dead.

Treatment.—A chrysarobin ointment should be applied (see formulæ at the end of this chapter).

Technique for Demonstration (*Montoya*).—1. Soak scales in ammonia for a few minutes.

2. Then place for five minutes in a saturated alcoholic solution of picric acid, to which 5 drops of acetic acid have been added.

3. Wash in distilled water and mount dry.

Note.—The picric acid hardens the mycelium and colours it yellow.

DHOBIE ITCH—Definition.—A tricophyton ring-worm, very common in the East, and showing a predilection for the crutch and surrounding areas.

Etiology.—Dhobie (or washerman's) itch is so called on account of its frequent connection with the native dhobie. *Manson* considers the belief as probably not very well founded that clothes can be contaminated by the washerman. But this is quite missing the whole point of Eastern procedure.

That clothes submitted to the usual vigorous rinsing, followed by an all too violent buffetting on the nearest stone, should thereby become infected, is, of course, highly improbable.

Perhaps some readers may have had occasion to travel in the East, and will have had (after a long voyage) about one and a-half days, or perhaps two days in port, before proceeding further. They will, perhaps, be struck dumb with astonishment to find that a native dhobie will bring back all their dirty clothes beautifully washed, dried, and starched in a matter of twenty-four to thirty-six hours. When these same people come to settle in the tropics and find that their washing, which then should be delivered weekly, is perhaps a day or two late even after the week is finished, they fail to remember their early experience with the native dhobie, and to put two and two together.

The fact is that the dhobie, to whose care we weekly commit our underlinen with child-like confidence, has a large clientèle. This Eurasian clerk or that Chinese krani want to go to a volunteer

dance; they have no dress shirt; their dhobie kindly supplies them—with yours. Or he wants a pair of superior white-drill trousers for a race meeting; his dhobie supplies him—with yours.

In this way are the garments of both sexes lent out; in fact, as many as three washes of one garment have been recorded during one week. Thus we can readily understand that our dhobie itch is not supplied by the dhobie himself, but by those customers who court fame in borrowed garments.

With regard to the causal fungus, *Castellani* considers that it is a tricophyton infection due to several different species, all of which are different from those found in Tinea circinata. *Manson* considers that many cases are produced by *Microsporon furfur* and *M. minutissimum*, and that they are really inflamed erythrasma. This is quite possible, although in none of the many cases observed by the author has there been any resemblance either to Pityriasis versicolor or to erythrasma.

The appearance and clinical features have been those of a Tinea circinata, and the microscopical features have been indistinguishable from Tinea ectothrix.

Symptoms.—The disease begins with a slightly raised red spot. This spreads by the periphery with an irregularly circular outline. The centre becomes clear, and all the activity of the fungus is concentrated on the spreading and festooned margin. This margin is red, raised, thickened, and generally rather moist. There is an intolerable itching.

When the ring has reached a diameter of 6 inches to a foot, it usually stops, or may coalesce with other foci.

The desquamation is extremely fine, and often scarcely noticeable.

The scratching demanded by the pruritus often leads to a condition of weeping eczema, and the whole scrotum, perineum, and inner thighs may be raw, and the patient suffer untold tortures.

Treatment.—This should be taken in hand early, and a chrysarobin ointment (see formulæ) applied promptly.

If the condition is not seen until a bad eczema has supervened, there is no remedy like glycerine of boracic acid, which often acts like a charm. It should be applied for a quarter of an hour, twice daily, on lint: during the intervals a little powdered boracic acid can be applied, and the parts kept from any contact or friction by lint and light bandage. The author has seen cases which have defied treatment for months yield to this treatment within a week.

RHIZOGLYPHUS PARASITICUS.—*Bell*, in Hongkong (1905), reports a case of infection with this animal parasite—one of the acarina.

It occurred in an Indian policeman who had been nine years in the colony.

The lesion consisted of a large circular eroded patch on the sole of the right foot, with a central irregular ulcer.

Itching was absent.

Some of the skin removed from the ulcer showed several of these mites.

Treatment with 4 per cent. formalin in glycerine was rapidly successful.

The condition is probably very rare.

PRICKLY HEAT—Synonyms.—*Lichen tropicus*, *Miliaria papulosa*, *Dysidrose sudorale*.

Definition.—An infection of the skin in which there is an obstruction to the sweat secretion due to inflammation (*Crocker*).

Description.—It consists of minute, bright red, acuminate, discrete papules, closely crowded together, with vesicles or vesico-pustules sparsely interspersed.

It comes out suddenly, preceded and accompanied by profuse sweating in other parts, and is attended with intolerable pricking and tingling (*Crocker*).

The condition may affect much or little of the body surface.

The parts most usually selected are the back, shoulders, forehead, and arms.

In consequence of scratching, dermatitis or boils may supervene.

In mild cases the disease may only last for a few days; or, on the other hand, it may continue till the advent of cool weather.

In a former patient of the author's—a ship's stewardess—the condition began at Port Said during each outward bound voyage to India. Her whole body was covered from head to foot, and no relief was experienced till the Mediterranean was again reached.

The treatment consists in avoiding flannel garments, iced drinks, or violent exercise.

Glycerine of borax is a useful application, especially if a little thymol be added.

CHEIRO-POMPHOLYX (*Hutchinson*)—**Synonyms.**—*Dysidrosis* (Tilbury Fox); *Pompholyx* (Crocker).

Definition.—A vesicular and bulbous eruption limited to the hands and feet, and connected with hyperidrosis.

The disease was originally described by *Tilbury Fox* and *Hutchinson*, independently, in 1875.

It is of rare occurrence. It occurs in the summer in cool climates, and is occasionally seen in the tropics.

The hands are the usual seat of the vesicles, more rarely the feet, and scarcely ever elsewhere.

The eruption commences with burning and tingling, followed by the formation of deeply embedded vesicles, singly or in groups, usually along the sides of the fingers and on the palms. These frequently coalesce into larger bullæ. The contents are neutral or alkaline, and slightly turbid in the older lesions. The bullæ may range from $\frac{1}{16}$ to 1 inch in diameter.

There would seem to be a connection with hyperidrosis. *Crocker* considers it of neurotic origin, "probably a vaso-motor neurosis

leading to inflammation in and about the sweat apparatus, but not limited to those structures."

In about a fortnight the contents become absorbed. The vesicles never rupture spontaneously. Recurring attacks are frequent.

The *treatment* should consist in the administration of internal tonics.

PELLAGRA (*Pelle agar* = rough skin)—**Definition.**—A disease of limited distribution, possibly of toxic origin connected with grain eating, and characterised by an erythematous exanthem.

Synonyms.—*Mal del sol, Risipola lombarda, Lepra italica, Scorbutus alpinus.*

History and Geographical Distribution.—It was first reported from Spain, by *Casal*, in 1735. Later, it spread over Italy and S. W. France. Besides these European centres, the disease is also endemic in Lower Egypt, Algiers, and Mexico.

Etiology.—Both sexes are equally liable to be attacked, and all ages may participate, although middle age is the most usual period.

It is very largely a malady of the poor, as unwholesome food and unhealthy surroundings predispose to the disease.

The condition is apparently an intoxication, and has been considered by many to be due to the continued eating of damaged maize. The reasons on which, according to *Scheube*, this conclusion is based do not seem altogether conclusive. They are—

1. That in those countries in which pellagra is endemic, maize is the staple food of the peasantry.

2. That extracts prepared by *Sombroso* from the damaged maize could produce symptoms resembling pellagra in animals and human beings.

Sombroso considers that the intoxication is caused by the product of a chemical change in the maize, due to a micro-organism which, *per se*, is harmless.

All the various fungi and bacteria found on maize have consequently been incriminated in turn, but no conclusive evidence has yet been brought forward to support the maize theory.

It has been said by many writers that the disease followed the introduction of maize into Europe. In answer to this it need only be said that nothing certain is known about the introduction of that cereal; and, moreover, it was certainly in dietetic use in Italy at least 200 years before the appearance of pellagra.

Symptoms.—Prodromal symptoms of languor and debility may be present for many months.

This is followed by anorexia, dyspepsia, and diarrhœa; and is accompanied by such nervous disorders as headache, vertigo, increased reflexes, erotic perversions, amnesia, and general debility.

At the time, or soon afterwards, an erythema of face, neck, chest, dorsum of hands and forearms supervenes.

These clinical features become ameliorated for a few months only

to be followed by exacerbations until after a few years serious cerebro-spinal symptoms are developed, such as—

Muscular weakness, paralyses, spastic gait, paræsthesia, increased reflexes, indolent pupils, cutaneous vasomotor disorders, loss of skin elasticity, melancholia, &c. Finally, a cachectic state is reached with marasmus, bedsores, &c., which finally kill the patient.

The *duration* of the disease may be prolonged to fifteen years or more. After the first stage of the disease the chances of recovery are small.

Post-mortem, the *liver* is enlarged and shows fatty degeneration; the *spleen* is small and atrophic; the *kidneys* are cirrhotic; the *gut* wall is thin and atrophied; the posterior and postero-lateral columns of the cord show a symmetrical sclerosis.

Chronic meningeal inflammation is frequent, and the cerebral cortex may be atrophied.

The peripheral nerves are normal.

The *treatment* consists in a generous and nourishing diet, together with the internal administration of arsenic.

ACRODYNIA (*Epidemic Erythema*).—This is a disease seemingly allied to pellagra and ergotism.

Epidemics have occurred amongst soldiers and prisoners both in Europe and tropical Mexico.

It would be of interest to be on the look out for the condition in other tropical regions.

The symptoms as given by *Crocker* are—

Gastro-intestinal irritation, injected conjunctivæ, facial œdema, palmar and plantar formication and prickings with a preliminary hyperæsthesia followed by anæsthesia.

Then comes an erythematous eruption, chiefly of hands and feet, but also occurring on the trunk, followed by exfoliation and dark pigmentation.

In severe cases there may be some marasmus and paresis.

There is no fever. Relapses are unusual. It is seldom fatal.

Recovery is usual in a few weeks or months.

There are no special *post-mortem* changes, and the pathology is obscure.

KELOID.—Keloids are unusually prevalent in yellow, black, and brown-skinned races, and are prone to follow slight injuries and abrasions or specific skin lesions.

One of the commonest forms seen amongst Chinese coolies is that following hypodermic injections of morphia. Of these the author has seen some 700 odd cases. The needle punctures have been followed by a raised scar keloid of about $\frac{1}{3}$ to $\frac{1}{2}$ inch in diameter. The whole arms are in some cases covered with such scars, and often the thighs as well, if the individual has been of very vicious habit.

The following two illustrations of entirely different types of keloid may be of interest.

To face p. 31[illegible].

To face p. 319.]

[Photo by the Author.

Fig. 95.—Keloid.—Type 2.

Keloid—Type 1 (Fig. 64).—A Hokkien, aged 28, had been a ckhsha-puller in Penang. Returned to Amoy two years ago. ad a sore on the penis seventeen months ago. About four months ter, a few itching red spots occurred on legs which wept after ratching. Quite gone in three months leaving pigmented scars, ither raised nor depressed. Present state shows considerable eas of back, arms, chest, and legs covered with an irregularly aped keloid growth. This was gradually disappearing from the gs upwards, leaving the pigmented areas referred to. His ther and mother died while he was a boy. He has one brother ho had the same affection some years ago.

His village contains some 300 inhabitants, and he knows of some n or more amongst them (including women and children) who ve the same complaint. The condition is possibly a syphilitic loid.

Keloid—Type 2 (Fig. 65).—A Hokkien, aged 34. Left enang four years ago for his native village near Amoy, since hen he has been a market gardener carrying tea. Condition gan three years ago with an itching spot on the back, which ept after scratching. More of such appeared in about ten days. he condition is limited to the back, and the keloids are discrete. few of them have been absorbed, leaving a slightly pigmented nd slightly raised area.

He knows of no one in his village with the same condition, nor ave any of his relations had it. There is no history of syphilis. Ie attributes it himself to the friction of wet shoulder loads.

A microscopical section kindly made by Dr. Keith of Singapore, owed only marked thickening of the corium, and no micro-ganisms.

VITILIGO—Synonyms.—*Leucodermia, acquired Leucasmus, eucopathia, Achromia, Piebald-skin.*

Definition.—An acquired disease characterised by the presence f symmetrical and progressive white patches with convex borders, irrounded by increased pigmentation.

Etiology.—The condition is fairly common amongst the dark d yellow races. The author has seen probably over 250 cases nongst Chinese coolies from Amoy, Swatow, and Hongkong (in ne year over 60 cases were counted).

Amongst the Malays it is not so common, although not infrequently een.

No case has been observed amongst the Indian immigrants to ingapore.

The condition has often been erroneously confused with albinism, hich is a congenital absence of pigment.

Vitiligo, on the other hand, is an acquired disease, and is almost ertainly a tropho-neurosis.

As a matter of diagnosis, it is not infrequently confused with *inta*, and illustrations of vitiligo shown to illustrate that disease.

The whitish patches of anæsthetic leprosy are said to resemble vitiligo, but it is rare that such a mistake could be made.

Symptoms.—An increased deposition of pigment precedes the white patches.

In the middle of this dark area a loss of pigment takes place, resulting in a milk-white patch. This patch enlarges symmetrically, driving the pigment before it.

The patches may be few or numerous, and may affect any or all regions of the body.

The hair turns white in the affected areas.

The disease takes many years to travel over the body.

There is no alteration in sensation or secretion, nor is there any subjective symptom, though pruritus occasionally precedes the onset.

No *treatment* has any controlling influence on the condition.

FORMULÆ.

Eczema.

℞.—Liq. carb. deterg.,	℥iss.
Hyd. amm. chlor.,	gr. xxv.
Lanolin,	℥ij.

M. Ft. ung.

Dhobie Itch.

℞.—Chrysarobini,	ʒi.
Ol. caryophylli,	♏x.
Ung. paraffin,	℥i.

M. Ft. ung.

(This is a specific for the earlier stages.)

Scabies.

℞.—Sulph. præcip.,	ʒiij.
Ol. limonis,	♏v.
Ung. paraffin,	℥xij.

M. Ft. ung.

Psoriasis.

℞.—Ichthyol,	5 parts.
Zinci oxid.,	5 ,,
Vernisol,	90 ,,

M. Ft. pigment.

(Keep well corked.)

Sig.—Smear a small quantity thinly over the infected part, and allow to dry.

(*Note.*—The film can be removed with a little warm water.)

Pityriasis versicolor or nigra.

℞.—Ichthyol,	. . . . . .	5 parts.
Sulphur,	. . . . .	5 ,,
Vernisol,	. . . . . .	90 ,,

(Keep well corked.)

Sig.—Smear a small quantity over the infected area, and allow to dry.

Cold Cream.

℞.—Adipis lanæ hydros.	. . . .	ʒi.
Ung. paraffin,	. . . .	ʒiij.
Ol. amygdalæ,	. . . . .	ʒiv.
Liq. calcis,	. . . . .	℥i.
Liq. plumbi fort..	. . . .	ʒss.
Ol. caryoph.,	. . . . .	♏v.

M. Ft. ung.

(Very useful for skin erythema.)

CHAPTER XXXII.

SMALLPOX.

Definition.—An acute infectious disease, endemic in the tropics, and characterised by pyrexia and a cutaneous eruption passing through papular, vesicular, and pustular stages.

Synonyms.—*Variola* (Latin), *La petite vérole* (French), *Blattern* (German), *Viruelas* (Spanish), *Vajuolo* (Italian), *Jadari* (Arabic), *Chachar* (Malay).

History and Geographical Distribution.—For a thousand years before the Christian era the disease was known, and inoculation practised in India and parts of China.

The first medical work on smallpox was that of *Rhazes* of Bagdad (tenth century), who wrote a treatise on it.

The first English work on it was by *Kellwaye* in 1593.

It is essentially an endemic disease of widespread tropical distribution, often assuming dangerous epidemic features, as in Calcutta (1906). Like the other cosmopolitan diseases—plague and cholera—smallpox has at one time or another visited epidemically large areas of temperate or cold climates.

Its first introduction into Europe was supposed to have been brought about by the Arabs in 710 A.D. at their conquest of Spain.

Serious outbreaks have occurred from time to time, such as in Iceland in 1241 and 1707; Sweden in the fifteenth century; the West Indies, 1507; London, 1871; but vaccination, combined with modern hygienic and sanitary principles (such as have been so effective in the case of plague and cholera) have succeeded in reducing its incidence to a minimum, and it is now chiefly, epidemically and endemically, confined to warm climates.

Etiology.—The causal agent of smallpox has occupied the attention of many observers, but up to the present with disappointing results.

In the later pustular stages many pyogenic organisms will, of course, be found, such as the *staphylococcus aureus* and *flavus*, and also many of the skin saprophytes.

The search for organisms in the earlier stages, as in vaccine lymph, failed to reveal any bacterial forms.

Attention was, therefore, directed to the quest of a parasitic protozoon; and a long list of observers, headed by Pfeiffer in 1887, claim to have found sporozoa.

These include *Guarnieri*, *Clarke*, *Ruffer*, *Plimmer*, *Ogata*, *Funck*, *Roger*, *Weil*, and recently *de Korté*.

It is almost certain, however, that these protozoa (if such) have ly a casual connection with the disease, for the following asons :—

(*a*) *Reed* has found amœboid bodies, apparently identical with ose of Pfeiffer, in the blood of normal children and monkeys.

(*b*) *Porter* has also found similar bodies in the blood of healthy eople.

(*c*) *Terroni* and *Massari*, by artificially inflaming the cornea with oton oil, obtained similar bodies, and therefore conclude that the -called parasites are derived from the nuclei or from emigrated ueocytes.

(*d*) *Love*, in the *Lancet* (1905), describes such bodies as having een found by various observers in the blood of patients suffering om measles, scarlatina, typhus, smallpox, and syphilis.

(*e*) Attempts at cultivation have failed.

(*f*) Observations on the mode by which the disease is spread; s conveyance by the atmosphere, by the agency of a third person, r letters, books, rags, &c., are all in favour of a schizomycetic igin rather than a protozoan.

Bacterial investigation has met with greater success.

Burdon Sanderson was amongst the first to demonstrate the esence of bacteria in vaccine lymph.

In 1896 Copeman and Klein, independently, discovered a acillus which is very possibly the specific cause of the isease. This has been named the *Bacillus albus variolæ* (Klein). *Klein* observed this organism in lymph from a calf's vaccine pustule n the sixth day; in human vaccine lymph on the eighth day; and a variolous pustule on the fourth day (*Muir* and *Ritchie*).

Attempts to cultivate this organism at first met with failure; but April, 1897, *Copeman* succeeded in cultivating the bacillus from oth sources in eggs, and from such egg-cultures was able to inoculate lves. *Klein* also, by storing variola scabs in 50 per cent. glycerine, nd so getting rid of extraneous saprophytic organisms, was able to ltivate this bacillus, morphologically identical with that observed vaccine lymph (*Hewlett*).

Both *Copeman* and *Kent* also found the bacillus in sections of accine pustules stained by Löffler's blue, or Gram. They occur in roups of three to ten both in the lymph and the tissues.

Technique.—Prepare films. Dry and fix. Place for five minutes in o per cent. acetic acid. Wash. Dry. Place in alcoholic gentian olet for twenty-four or forty-eight hours. Wash, dry, and mount.

Characteristics.—B. albus variolæ—

(*a*) Rod-shaped; 0·4 to 0·8 μ in length and 0·2 μ in thickness.
(*b*) Apparently same group as *B. diphtheriæ.*
(*c*) Stains better at poles than centre.
(*d*) Non-motile.
(*e*) Does not liquefy gelatine.
(*f*) Stained by Gram.

(*g*) Small, white, opaque, coherent colonies on agar.
(*h*) Feeble growth on gelatine.
(*i*) Involution forms occur.
(*j*) In centre of protoplasm is often a clear globule, thought to be a spore.

Until further developments, therefore, we may tentatively ascribe the disease to infection by this "*B. albus variolæ.*"

Susceptibility.—All races are liable to attack, especially the negro (*MacCombie*).

Attacks are slightly more common amongst males than females.

With regard to age, the incidence rate in early infancy is rather low, though many cases do occur, and even may occur *in utero*, as in *Warner's* case where two fœti were born with a smallpox eruption shortly after the recovery of the mother from an attack. Youth and middle age are, *par excellence*, the period for smallpox. It is rare after the age of fifty, and extremely rare after sixty. In nearly 300 cases seen or treated by the author, there has been no instance of attack after that age.

Epidemiology.—The following are the methods of recognised communication:—

1. *By patients.*—The incubation stage is probably not infectious. The stage of initial fever, and the first two days of the rash seem to be slightly infection-bearing.

Infection is virulent during vesiculation and pustulation; and very virulent during desquamation.

Infection may be contracted either at close range or at a distance of several yards according to the ventilation of the apartment. Severity of an attack thus contracted will depend on the susceptibility of the visitor, rather than on the gravity of the patient's condition.

2. *By smallpox corpses.*—That infection may be so carried is well recognised. Antiseptic wrappings should be applied immediately after death, and careful attention paid to the proper sealing up of the coffin.

3. *By a third person.*—Attendants or visitors may convey the infection to others, although not attacked themselves. This is probably carried by some part of their person.

4. *By various articles.*—Bedding, furniture, rags, clothing, coins, &c., may all carry the infection.

An epidemic with 34 fatal cases was originated by an infected letter in Saginaw, Michigan, in 1906.

One of the greatest examples of international ineptitude is found in Article 16 of the Paris Convention of 1903, which exempts letters and mail from disinfection or restriction. Both smallpox and cholera can be thus conveyed, and, possibly, plague.

5. *By the atmosphere.*—That it is conveyed by the atmosphere is now well established.

A non-immune person entering an infected room and leaving immediately, will often contract the disease (*vide* method 1). This

probably due to atmospheric particles infected by the bacillus or its spores.

In the same way propinquity to a smallpox hospital ensures a high incidence amongst the neighbouring inhabitants, which diminishes as the radius extends until it reaches zero at a distance of nearly three miles (*Thresh*). This has been shown in many places, notably by *Power* at Fulham; by *Barry* at Sheffield; and by *Thresh* at Purfleet.

6. *By inoculation.*—This was practised in prehistoric times both in India and China. It even had considerable popularity in Europe during the eighteenth century. Virus was taken from a pustule of a mild case of smallpox and inoculated by scarification. In the majority of cases a mild form of the disease was thus conveyed and conferred subsequent immunity. Its disadvantages were that the disease so conferred was still contagious, and might be a focus for a virulent outbreak.

Jenner's discovery of vaccination provided a safe substitute, and the practice is now discontinued in civilised countries, although frequently practised still by natives. This practice contributed nearly 3,000 cases out of 3,899 cases at Brunei (in N. Borneo) in 1904.

Method of Infection and Pathology.—An elaborate paper by *Stokes* in the *Bulletin of the Johns Hopkins Hospital*, 1903, indicates the probable method of infection.

The bacillus or its spores are inhaled, and the primary infection is in the lungs.

The poison then enters the circulation, and shows a selective partiality for the epithelium of the skin and respiratory tract.

The serious and fatal lesions are caused by the *secondary* infection from the skin and respiratory tract, the agent usually being *S. pyogenes*. The distribution of this organism throughout the lesions well explains most of the pathological changes, such as the pulmonary condition, thromboses and local necroses.

Lesions of the viscera are seldom found. Pericarditis, endocarditis, peritonitis, nephritis, and myelitis are all rare.

Pyæmic or septicæmic manifestations may be found in severe cases.

It is in the respiratory passages, however, that the most marked pathological conditions occur:—Rupture of pustules in the palate or pharynx may give rise to ragged ulcerations and stomatitis. Vesicles on the mucous membrane of the larynx will give rise to laryngitis. Congestion of the lungs and bronchitis are universal in severe cases. Catarrhal pneumonia is exceedingly common in confluent and hæmorrhagic cases, and is a frequent cause of death in children. Lobar pneumonia is fairly common in pustular stages, as also is septic pleurisy.

Symptoms.—After infection there is an *incubation period of twelve to fourteen days* before the first clinical symptoms manifest themselves. Various observers have given from five to twenty-one

days as the incubation limits; but, as in the case of so many other infectious diseases, it is generally impossible to point to a definite day as that on which the infection was contracted, as the sources of infection may be numerous. In those cases in which errors are excluded, it will usually be found to be either twelve or fourteen days.

The incubation being concluded, the onset is, as a rule, sudden.

There is severe frontal headache, aggravated by movement. Severe lumbar and sacral pain is almost always present, and the temperature begins to rise.

There is anorexia and thirst; the breath is often fœtid, and the tongue furred. Vomiting is frequently an early, and, with children, a very usual, symptom. The urine is scanty, and contains urates.

The pulse is rapid, but is full, strong and regular.

The temperature usually attains its maximum about the second day. These initial symptoms continue until the appearance of the *eruption*, which occurs *on the third day*.

It has been stated by some that smallpox can occur without an eruption, but this is highly hypothetical.

There are three clinical types of smallpox:—

1. The discrete, or modified;
2. The confluent;
3. The hæmorrhagic;

and each of these may vary in severity.

1. *The Discrete and Modified.*—On the third day of the fever the characteristic eruption occurs, and with its appearance the temperature (which had then reached its maximum) begins to fall, and the urgent symptoms of headache and backache to abate.

For a few hours the eruption is macular, but quickly develops into small, red, raised, shotty papules—seen first on the forehead and face, and becoming generally developed some hours later.

Throughout the disease, however, they are most numerous on the forehead, face, hands, wrists, ankles, and feet.

The papules are also distributed over the buccal mucous membrane, and are often present in the larynx and trachea. Not infrequently they are to be seen in the vulva and vagina.

After the progress of some hours, they are seen to be surrounded by a thin red areola.

By the fifth day of the eruption the papules have become vesicular, and attained their maximum limit of growth. This is usually accompanied by slight subcutaneous œdema of face, neck, hands, &c. There is a feeling of much discomfort, and some thirst.

By the eighth or ninth day of the eruption, the vesicles have become pustular, having an opaque appearance, and usually surrounded by a vivid areola.

In discrete cases (Fig. 67) there is now a secondary rise of tem-

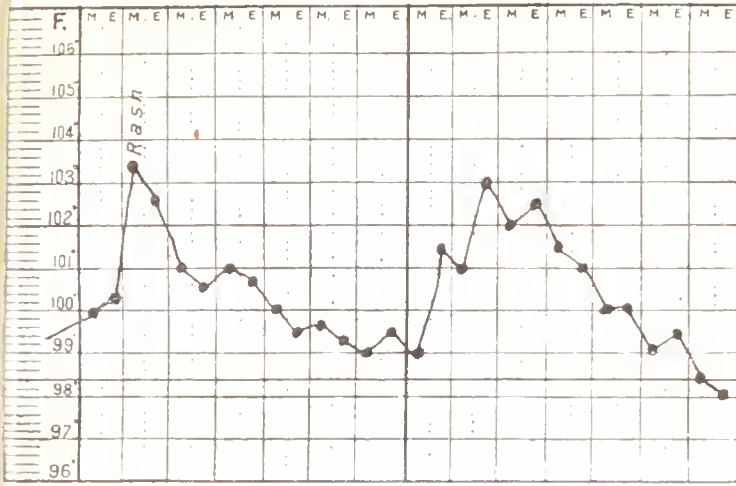


Fig. 66.—Severe, discrete smallpox. Recovery.

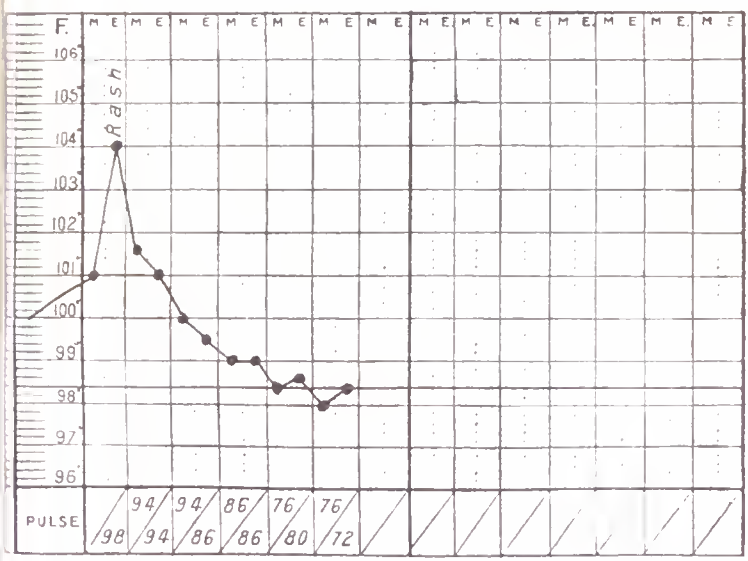


Fig. 67.—Modified smallpox. European. Aged 27. Recovery. (Discharged in four weeks. Note absence of secondary rise as often found in modified cases.)

perature, which may reach 102°, 103°, or 104°. This secondary rise is due to the secondary pyogenic infection.

In cases modified by previous vaccination (Fig. 67) there is very often no secondary rise at all, and the pustules may dry up soon, without attaining a large size or becoming angry.

The pulse rises during this pustular stage, and there may be some mental confusion, though delirium is rare in discrete cases.

The raised temperature, after persisting for one or more days, falls, as the pustules begin to desiccate, until it reaches normal on about the tenth or twelfth day of the eruption.

Desquamation will generally be finished in about three or four weeks.

In these discrete cases there are rarely any complications or sequelæ, and pitting is not very marked; in fact, in modified small-pox it may be absent.

2. *The Confluent.*—All degrees of confluence are met with. It may be limited to the face or extremities (Fig. 68), or may be widespread.

The commencement of an attack is much the same as in discrete cases. Shortly after the eruption is declared, however, considerable erythema of forearms, face, neck, and perhaps elsewhere, is manifested.

The irritation and discomfort become very marked. The vesicles gradually coalesce, and as the pustular stage approaches, the subcutaneous œdema becomes very great. The eyes and mouth can scarcely be opened, nor the fingers moved.

The scratching demanded by the intense irritation leads to raw legs and arms and face; pus and sero-sanguinous fluid exudes; a peculiar and foul fœtor emanates from the patient; and the whole clinical picture is one of the most concentrated hideousness.

In about 50 per cent. of these cases the secondary temperature rises high; the pulse rate rapidly increases; a condition of septicæmia supervenes, with muttering delirium, relaxed sphincters, diarrhœa, increasing coma, and finally death, which usually occurs from the tenth to the fifteenth day of the disease (Fig. 69).

In the other 50 per cent. of confluent cases, as the crust formation occurs, there is a gradual, but regular, fall in the fever and the pulse rate; the subcutaneous œdema slowly subsides; the raw surfaces begin to heal; and, finally, at about the third week the stage of convalescence is entered on.

3. *The Hæmorrhagic.*—This almost invariably fatal form is somewhat polymorphous in its clinical manifestations.

It is characterised by hæmorrhages, both cutaneous and internal.

The initial symptoms are generally exaggerated. Vomiting is very often a prominent feature.

The backache and headache are very severe.

The temperature may mount as high as 103°, but rarely higher. On the first or second day of the fever, a vivid erythema becomes generalised, developing into red or purple petechiæ in various places.

[*To face p.* 328.

Fig. 68.

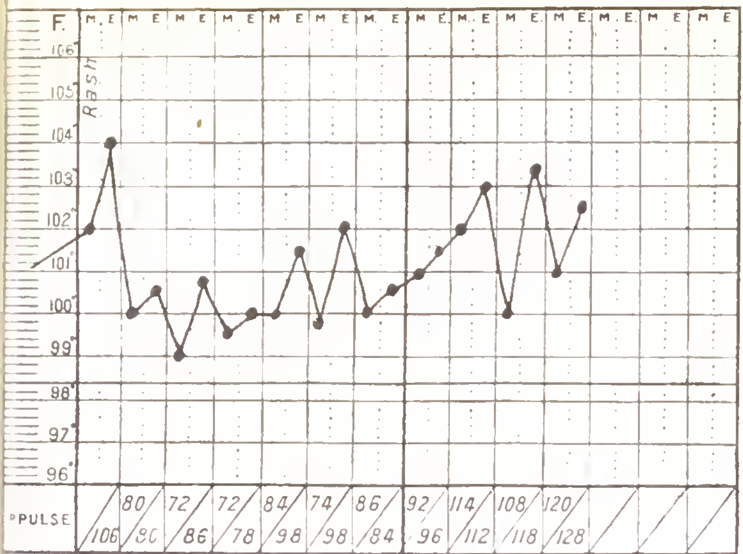


Fig. 69.—Confluent smallpox. Male European. Aged 32. Death. (Not vaccinated since infancy.) *Case from the Author's practice.*

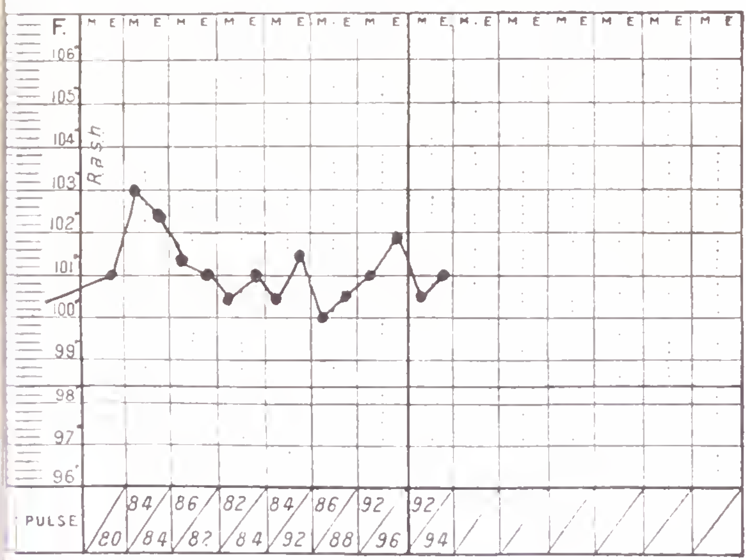


Fig. 70.—Hæmorrhagic smallpox. Indian Male. Aged 20. Death.

The conjunctivæ are suffused. Epistaxis and hæmoptysis may occur, and there is oozing from the gums.

On the third day of the fever these symptoms are complicated by the development of the characteristic rash.

In a very small percentage of such cases, perhaps only half-a-dozen or so of isolated papules appear. Purple and violet-ink spots are found on face and limbs; conjunctival and retinal hæmorrhages may occur; the temperature falls to normal, or even subnormal; there is rarely any mental obscurity or delirium; but the pulse is soft, and, in a more or less collapsed condition, death takes place from syncope between the third and sixth day. Such cases are sometimes called black smallpox.

In the more usual form of hæmorrhagic smallpox, the characteristic eruption is general. Hæmorrhages occur, both subcutaneous, intra- and peri-papular. The temperature rarely falls below 100° F. (Fig. 70). There is usually delirium. Sordes collect round the mouth. There is considerable subcutaneous œdema as well as hæmorrhages. The pulse is quick, and of low tension. Respiration is hurried and shallow. Secondary septicæmia usually ends the chapter, between the seventh and tenth day of the disease.

Diagnosis.—In the pre-eruption stage, it is, of course, highly difficult (except during epidemics) and often impossible, to diagnose the disease.

During the late vesicular and pustular stages there should be no difficulty in diagnosis.

In the early stages of the eruption, however, it is often a matter of doubt as to whether a case is one of chickenpox, or of mild or modified smallpox.

The following differential table may perhaps be of use in the diagnosis:—

Chickenpox.	Smallpox.
Incubation period may extend to sixteen days.	Incubation not more than twelve to fourteen days.
Rash appears on first day of fever.	Rash on third day of fever.
Initial symptoms usually absent.	Headache and lumbar pain usually pronounced.
Distribution of eruption most abundant on the trunk, less on face, and least of all on hands and feet. (Figs. 71 and 72.)	Rash first appears, and is most abundant, on face, forehead, and extremities.

Chickenpox.	Smallpox.
Temperature only slightly raised before rash and does not usually fall with its appearance.	Initial temperature often high and falls with the appearance of the eruption.
Eruption becomes vesicular in a few hours.	Eruption not vesicular for twelve hours or more.
Many of the vesicles are oval. (Fig. 72.)	Vesicles are all round.
Vesicles when pricked collapse.	Vesicles do not collapse when pricked.
Vesicles when circular are hemispherical and dome-shaped.	Not until pustular stage are the lesions hemispherical and dome-shaped.
The diagnostic characters of the vesicles are most marked on the trunk. On the extremities they may show greater resemblance to smallpox.	
Vesicles fully developed in one day.	Vesicles fully developed in five days.
The eruption may continue coming out in crops for several days.	The eruption comes out at once, though its appearance on the face and extremities may precede that on the rest of the body by several hours.

In rare cases mistakes in diagnosis may occur in other directions than that of chickenpox.

Two instances which occurred to the author during the week preceding the writing of this chapter may be quoted as examples of smallpox simulating plague and measles.

A Chinese boy arrived at Singapore with 1,900 other coolies. He had been ill for three days before arrival, complaining of giddiness. There had been no headache or backache. He had had no fever until the evening before arrival, when it was 99·5. When seen by the author he had a temperature of 103·5. He had previously had some epistaxis. His gait was reeling and drunken. His tongue furred white, with red tip and edges. His eyes were suffused, his expression anxious. In fact, in every particular he gave the impres-

sion of plague. Neither pneumonia nor gland enlargement was to be found, and, therefore, with the idea that it was septicæmic plague, his blood was examined for the bacillus. As this gave a negative result, he was removed to the quarantine station for observation. The following day he was delirious, and his pulse and appearance were even more suggestive of plague than before. On the third day, however, a typical smallpox rash appeared, and the diagnosis was cleared up.

The second case was that of a coolie removed from a batch of quarantined contacts to the hospital for treatment. He had lachrymation, conjunctival suffusion, some pyrexia, and a typical raised, velvety, morbiliform eruption of measles over neck and trunk. Three days later the typical measles had developed into an equally typical hæmorrhagic smallpox.

Treatment.—As there is no specific remedy, everything depends on symptomatic treatment; and in this disease, as much as any, the scale may often be turned in the right direction by careful nursing and unremitting attention.

The sick room should be airy and well ventilated; the bedding soft and light. It is as well to cut the hair short early in the disease.

In the early stages, milk or milk and soda is all the nourishment necessary. In the later vesicular and pustular stages, more nourishment should be given, in the shape of Valentine's beef juice, custards, and iced jelly. Ice to suck is very grateful, both in quenching the thirst and soothing the inflamed buccal mucosa.

In all severe cases, alcohol is urgently indicated from the beginning of the pustular stage. It is best given as brandy, mixed with milk and iced, in frequent small doses.

Iced champagne may be given as an alternative.

As regards the drug treatment, it is unwise to give opium. The cardiac depression, which is such a feature of serious cases, is only aggravated by the administration of this drug.

In the painful, irritable, and often delirious stages, the safest and promptest effect is generally produced by giving a hypodermic infection of hydrobromide of hyoscine, gr. $\frac{1}{100}$, and repeated in doses of gr. $\frac{1}{200}$ if or when necessary.

Local applications, as a rule, are unnecessary during most of the disease. If, however, bed-sores form, they should be treated by zinc oxide dressings. For the raw and denuded epidermis an ointment made of boracic acid and lanoline should be continuously applied on lint. During the desquamating stage daily warm antiseptic baths should be given, and followed by the application of carbolic oil.

The condition of the eyes should be carefully noted. Conjunctivitis, keratitis, and corneal ulceration are frequent accompaniments of the disease. It is, therefore, highly necessary to see that the eyes are kept free from discharge. The lids should be oiled to prevent sticking.

[To face p. 332.

Fig. 71. Chickenpox. Chinese Woman. Front View.

To face p. 333.]

Fig. 72.—Chickenpox. Chinese Woman. Back View.

Weak boracic lotion should be used to wash out the conjunctivæ. For keratitis, atropine drops should be employed; or, if there is increased tension, iridectomy may be necessary. For corneal ulcer, an ointment of hyd. ox. flav., gr. i. to the dram, should be used.

Nursing should be most carefully carried out. Frequent changing of the much be-fouled bed-linen will be necessary.

Constant watching is necessary during the delirium. Bed-sores should be looked out for.

The regular and careful administration of nourishment and stimulants should be unremittingly carried out.

The condition of the mouth should be frequently observed and attended to.

Convalescence will demand generous diet, with tonics and stimulants.

Reason would suggest that the administration of large doses of polyvalent anti-streptococcus serum, during an early stage of the disease, would be followed by beneficial results in curtailing the secondary pyogenic infection which causes the serious and fatal lesions.

The treatment by *red light*, which was first mentioned by John of Gaddesden in the fourteenth century, has been recently revived by Finsen. He maintains that if patients are placed in red light during the first day or two of the disease, and kept there until the pustules have dried up, that secondary fever will be prevented, and suppuration of the vesicles avoided.

A trial of the method, however, by *Ricketts* and *Byles*, at the smallpox hospital of the Metropolitan Asylums Board, led to the conclusion that "the mental symptoms were aggravated, the suppurative fever higher, and the septic sequelæ more numerous."

VACCINATION.—Inoculation, which had long been practised, had the disadvantage of conveying a disease which, although mild, was still infectious.

At the close of the eighteenth century there was a popular belief that cow-pox contracted from an infected animal, conferred immunity from smallpox.

There is a disease of the horse, known as "horse-pox" or "grease," which consists of ulceration in the region of the hocks.

Jenner showed that matter from this horse-pox could give rise to cow-pox in cows by manual transmission to the teats. A papular eruption on the teats was the outcome, becoming afterwards pustular, and then scabbing.

Jenner noticed that abrasions on the milker's hands, if infected by a cow-pox teat, gave rise to a local eruption, with fever, malaise, and anorexia.

He next showed, experimentally, that persons who had gone through such an attack, did not react to inoculation with smallpox; and, moreover, immunity was also secured in persons to whom cow-pox was artificially communicated.

Such was the inception of protective vaccination.

There were, at first, many opponents, but it has gradually gained almost universal confidence.

Jenner's observations and experiments were published in 1798, under the title, *An Inquiry into the Causes and Effects of the Variola Vaccinæ.*

The reduction in the London smallpox mortality, since the gradual introduction of vaccination after the year 1800, is well shown by the following table :—

Years.	Average Annual Deaths per Million from all Causes.	Average Annual Deaths per Million from Smallpox.
1660 to 1679, . .	80,000	4,170
1728 ,, 1757, . .	52,000	4,260
1771 ,, 1780, . .	50,000	5,020
1801 ,, 1810, . .	29,200	2,040
1831 ,, 1835, . .	32,000	830
1838 ,, 1853, . .	24,900	513
1854 ,, 1871, . .	24,200	388
1872 ,, 1882, . .	22,100	262
1883 ,, 1892, . .	19,800	73

CHAPTER XXXIII.

SPIRILLAR FEVER.

Definition.—A disease of warm and temperate climates, characterised by alternate periods of pyrexia and apyrexia, and caused by the presence of a blood spirillum.

Synonyms.—*Relapsing fever*, *Famine fever*, *Tick fever*.

Note.—*Novy* and *Knapp* have expatiated on the plurality of relapsing fevers on account of some slight morphological differences in the spirochætes found respectively in relapsing and tick fevers.

For clinical and didactic purposes *malaria* is considered as an entity, although at least three different forms of hæmamœbidæ are the causal agents in its production. In the same way, small morphological differences in the spirilla of relapsing and tick fevers are not sufficient ground for differentiation of these two diseases, which have so much in common. They are, therefore, both dealt with in this article under the heading of "Spirillar Fever."

History and Geographical Distribution.—*Hippocrates* described an epidemic of a relapsing fever in the island of Thasos, which was probably identical with our spirillar fever.

We then hear nothing about it through the long centuries until *Rutty* in 1770 described it as one of the diseases found in Dublin. It was then doubtless often confused with typhus and enteric, but *Henderson* of Edinburgh in 1843 argued that it should be considered as a separate entity. It was not until 1873, however, that *Obermeier* (assistant to Professor Virchow in Berlin) demonstrated the presence of spirilla in the blood of patients suffering from the disease.

Carter, working in Bombay in 1879 (where the disease had been brought in 1877), verified the observations already made in Europe, and concluded it as certain that the spirilla were the causal factor in the fever, because (1) after an interval blood infection is always followed by fever; (2) spirilla increase with advent and rise of fever, although there is no constant relation between the number of organisms and the intensity of fever; (3) the organisms disappear during the apyrexia; (4) blood containing the organisms can convey the fever, *Koch*, *Carter*, and others having thus produced the disease in monkeys.

There have been epidemics in Europe, 1818, 1843, &c., and elsewhere, Its presence was demonstrated in India and Egypt, but little research work was undertaken in other tropical countries as to the presence or absence of a spirillar fever, until *Dutton* and *Todd* discovered on 26th November, 1904, independently of *Ross* and

Milne, that a blood spirillum was the cause of a fever common in the Congo Free State, and there known as Tick fever. There are a few morphological differences between this spirillum (*Spirillum duttoni*) and that of the older recognised relapsing fever (*Spirillum obermeieri*), but the type of relapsing fever produced is in both cases almost identical.

Since then, in 1906, *Cole* has reported a case of spirillar fever at Ningpo in China; *Cropper* in Palestine; and *Wellmann* in Angola (West Africa).

It would seem, therefore, to be highly probable that the disease has a wide tropical distribution.

Etiology.—The following is a tabular comparison between the two organisms which cause spirillar fever:—

	S. obermeieri.	*S. duttoni.*
Length, . . .	8 μ (*Novy & Knapp*). 16 to 40 μ (*Kanthach*). *Allbutt's Medicine.*	16 μ (*Novy & Knapp*). 13 to 40 μ (*Stephens*). *Medical Annual.*
Number of spirals in single cell,	2 or 3.	2 or 3.
Width of spirals, .	1 μ.	2 to 2·7 μ.
Shape, . . .	Usually curled at ends. Frequently in loops.	Figure of 8, or circular tendency.
Flagella, . . .	Single whip-like flagellum at one end; equal in length to cell itself.	Diffuse flagella.
Animal susceptibility,	Benign infection of monkeys, rats, mice, rabbits.	Malignant infection of monkeys, rats, mice, rabbits. The dog and horse have also been infected (*Breial*).
Serum, . . .	Immunising serum has been prepared. It does not protect against *S. duttoni*.	Immunising serum has been prepared. It does not protect against *S. obermeieri*.
Intermediate hosts, .	Unknown. Probably fleas, bugs, or mosquitoes.	A tick (*Ornithodorus moubata*).

There is some question as to the *nature of the Spirochætes.*

Until 1904, it was generally assumed that the spirillum of relapsing and other pathogenic spirilla were bacterial in nature; but Schaudinn's remarkable work on *S. ziemmani* seems rather to point to their protozoal nature.

According to *Schaudinn*, the morphology of the spirochæte is transient, and marks a developmental stage of a trypanosome. He claims to have traced their development in the blood of an owl, *Athene noctua*, and in the body of *Culex pipiens* through an ookinet and trypanosome stage.

Koch has failed to corroborate this view. He has traced the spirochæte through the stomach, body, and ovaries of the tick, into the eggs, where he detected them in about every fifth egg. Thenceforward the spirochætes continued to increase in number and become aggregated into masses. The embryo tick is thus infected.

The following seven points have been adduced to negative their protozoal origin:—

1. *Koch* has found evidence of transverse division.
2. Both living and stained specimens fail to show any indication of a nucleus, blepharoplast, or undulating membrane.
3. The very rapid multiplication in the blood of rats (in half the time occupied by trypanosomes) inclines to bacterial habit.
4. When dialyzed against running distilled water, spirochætes remain motile for twenty or more hours (as do cholera spirilla), whereas trypanosomes become non-motile in less than two hours.
5. When heated to 45°, trypanosomes are killed, and disappear in less than half an hour; the spirochætes remain alive for a longer time; and, when dead, do not change their form.
6. Immune serums are not readily produced by protozoa and have a weak action.

Trypanosomata or piroplasmata serum has a prophylactic, but very little germicidal or curative effect.

With spirochæte serum a high degree of immunity can be secured, comparable to that which can be obtained with the cholera germ.

7. Trypanosomes in culture show a marked avidity for air, which is not shared by spirochætes.

The nature of the spirilla must, therefore, be considered as still *sub judice.*

Staining of Spirilla—1. Any aniline stain may be used.

2. The organism does not retain Gram's stain.

3. For examining details Schaudinn's syphilis stain should be used (see Chap. xxxix.).

4. For observation of flagella, Pittfield's flagella stain should be used (see Chap. xxxix.).

Age and Sex Distribution.—No race or sex is exempt. The disease may occur at any age, but is most common between the ages of 15 and 25.

Bad sanitation and food, and overcrowding are all *predisposing causes*, not only because of the lessened vitality and resistance induced thereby, but because the bed-bug, house tick, or other intermediate host is thus more easily able to increase both the supply and demand of his unknown parasitic guest.

One attack does *not* confer immunity.

Symptoms.—There are three clinical types of spirillar fever—

1. The usual or typical course.
2. The typhoidal course, with jaundice.
3. The uræmic course, with anuria.

1. *The Typical Course.*—After an *incubation period* of one week or under (three days in the case of direct inoculation) the disease sets in fairly suddenly with no prodromata. A little giddiness and headache together with a rigor may usher in a pyrexia which rises somewhat quickly to 104° or 105° F. (Fig. 73). The temperature remains high for 3, 5, 7, or 9 days—generally, rather towards the smaller figures if the parasite is *S. duttoni*—during which period there is headache, backache, and much aching of the bones. Vomiting is not infrequent, and occasionally diarrhœa may be present. Prostration and depression are intense. The skin is hot and burning; the patient is restless and flushed—pulse and respiration are accelerated. The skin is yellowish; the splenic dulness is increased, and usually also that of the liver.

After this condition has been maintained, usually for either five or seven days, a crisis suddenly occurs. A copious diaphoresis, often ushered in by a rigor, and sometimes accompanied by diarrhœa or epistaxis, contribute to a sudden fall of temperature and pulse rate. All the accompanying symptoms disappear and the patient feels quite well. Such a crisis nearly always occurs at night.

The crisis is followed by an *afebrile interval* varying in length from five to nineteen days, more usually about seven to nine days.

At the end of this time a *relapse* occurs. The onset of the relapse will usually be found to occur somewhere about fourteen days from the first commencement of the illness. The fever returns, and all the urgent symptoms are repeated. Pregnant women generally abort during this first relapse.

The duration of this relapse is not as long as the first attack, and is usually over in three days, again ending by crisis as before, and leading usually to the establishment of complete convalescence.

Occasionally, however, a second or third relapse may occur, with gradually lessening virulence, until convalescence begins.

2. In the *typhoidal type*, diarrhœa is the prominent symptom, the crisis is delayed, and there is much delirium. Hepatic enlargement is marked, and tenderness over the liver is often great. Petechiæ and hæmorrhages are not uncommon.

3. In the *uræmic type*, the secretion of urine is diminished; anuria and uræmic coma supervene. Both these types are uncommon.

Prognosis and Diagnosis.—The mortality will naturally vary with the personal resistance of the patients, as well as the gravity of type of disease in epidemics or districts.

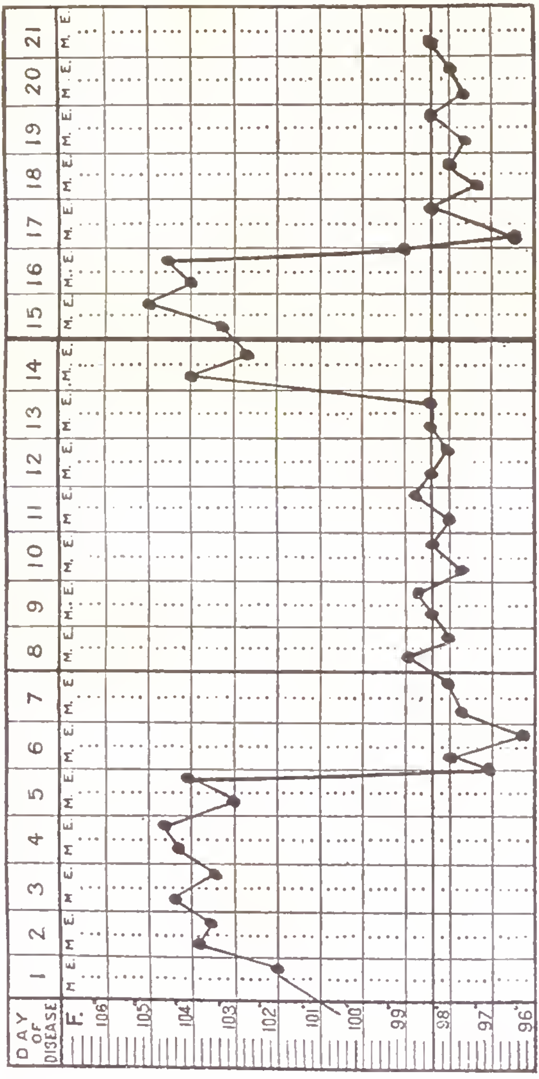


Fig. 73.—Type of temperature chart in a typical case of "spirillar fever."

As a rule, the death rate of the affected is from 4 to 10 per cent. In adverse circumstances it may rise as high as 50 per cent.

The prognosis is therefore, as a rule, favourable; but should be guarded if severe hæmorrhages, excessive delirium, or scanty urine supervene.

In diagnosis it may be necessary to exclude typhoid and typhus fevers, Malta fever, kala-azar, malaria, influenza, or dengue.

The sudden onset and the crisis in five or seven days after a continued and fairly high temperature should establish the diagnosis, made certain, of course, by the finding of spirilla in the peripheral blood.

Morbid Anatomy.—The *heart* muscle is pale, flabby, and friable.

The *spleen* is enlarged, softened, and fatty. Congestion of the vessels and increase of cell elements contribute to the swelling of the spleen pulp. The spirilla, during apyrexia as well as pyrexia, are present in the spleen in large numbers, where they are mostly enclosed in cells. These cells appear to undergo degeneration, and possibly the released spirilla may thus contribute to the relapse.

The *liver* is enlarged to some extent. A cloudy swelling of hepatic cells and small celled infiltration along the portal vein may be made out; spirilla may be found in the cells, and subsequent disintegration lead to foci of necrosis.

The *kidneys* are enlarged. The parenchyma is soft and flabby. The tubuli are fatty, and their lumen filled, partly with hæmorrhagic clots, and partly with a transparent fibrinous material. The interstitial tissue shows small celled infiltration.

The *lungs* are occasionally hepatised, or the bronchi inflamed. They are usually congested throughout. The *bones* show reddening of the marrow, and the parasite may be found in the marrow cells.

The *blood* does not contain spirilla except for some hours or days before the crisis; after which they disappear, and remain absent during the apyrexial intervals.

Sequelæ are not common. Recovery is usually complete with the termination of the attack. As with so many other infectious fevers, parotitis is sometimes found.

Treatment.—Quinine is useless and no drugs will modify the course of the disease.

Pain may be treated by the cautious administration of opium; headache by antipyrine; the distressing vomiting by sucking ice or applying sinapisms to the epigastrium.

Probably serum therapy will do much in the treatment of the disease; but, so far, not much work has been attempted in this direction.

CHAPTER XXXIV.

SPRUE.

(From the Dutch word *Spruw.*)

Definition.—A disease of warm climates tending to become chronic, and characterised by (1) a bare and usually painful tongue; (2) frequent attacks of buccal ulceration; (3) frequent soft, bulky, fermenting and pale stools; and (4) anæmia and debility.

Synonyms.—*Tropical aphthæ, Psilosis linguæ, Diarrhœa alba,* and possibly "*Ceylon sore mouth,*" and "*Indian hill diarrhœa.*"

Geographical distribution.—It occurs in the Malay Archipelago, in India and Further India, in Ceylon, in South China, and the West Indies.

It was first described by *Hillary*, in Barbadoes, in 1776.

Etiology.—The specific cause is not known. Both the small nematode *Rhabdonema intestinale* and the *Amœba coli* are often to be found in the stools of sprue patients, but are in no way responsible for the disease.

The chief *predisposing causes* are:—Prolonged residence in the endemic area, and exhausting conditions, such as dysentery, hæmorrhoids, frequent child-bearing, syphilis, bad food, &c.

The disease never appears epidemically, and is (according to *Scheube*) certainly not contagious. *Galloway* (of Singapore) in more than 25 out of 104 cases has, however, traced an indirect contagion.

The disease is found amongst natives, but 85 to 90 per cent. of cases occur in *Europeans* who have lived long in the tropics; and the first symptoms may not appear until months or even years have elapsed after leaving the endemic region.

It is confined to no sex or age, but middle-life contributes the most cases, and it is more common in women than in men.

The use of highly-seasoned dishes possibly plays some part in predisposing towards the condition, as also the abuse of alcohol so common in the tropics.

Symptoms.—The onset is, as a rule, insidious. The first symptom is usually some epigastric fulness or uneasiness, accompanied by eructations and heartburn. This continues for some months, the appetite being indifferent, and the stools irregular—constipation alternating with a morning diarrhœa.

The mouth then begins to get sore. Small raised red spots appear on the edges and tip of the tongue, and gradually coalesce

until the whole tongue has a red shining appearance as if varnished. A small ulcer (Crombie's molar ulcer) is frequently observed between the last two molar teeth in the upper and lower jaws.

The patient begins to get emaciated and anæmic; the stools become pale, frothy, and fermenting, and are passed in copious quantities daily. The appetite continues to fail, and not infrequently food is vomited. The mouth condition becomes aggravated. The erosion of mucous membrane of the lips, cheeks, pharynx, and uvula spreads, and may even involve the palate. Speech is in consequence painful and difficult; and any hot, sour, salted or seasoned dishes cannot be endured.

There is a feeling of physical weakness, loss of memory, and irritable temper.

The skin becomes muddy, the liver is found to be atrophied, the urine is diminished, and has a heavy sediment of urates.

Under treatment, there may be alternate aggravations and improvements.

In the above clinical picture of a subacute case, the disease may last for one or two years, when the patient will succumb to exhaustion or to intercurrent disease.

On the other hand, it may become chronic, and drag on for ten or fifteen years.

The clinical features in various cases may vary considerably.

Thin differentiates three forms of sprue:—1. *Buccal symptoms most pronounced* (occurs chiefly in Malayan type).

2. *Buccal symptoms late* (occurs in Indian type).

3. *Very chronic type* (observed in elderly people and oldest residents).

Manson notices six types:—1. *Protopathic Sprue.*—An ordinary attack of sprue, untreated or mismanaged.

2. *Secondary to Dysentery.*—Dysenteric stools gradually change into those of sprue. The mouth at the same time becomes sore, and soon a typical sprue is established.

3. *Secondary to Entero-colitis.*—Commences as an attack of febrile, acute, colicky diarrhœa, and gradually assumes the features of sprue.

4. *Gastric Cases.*—An incomplete type affecting only a limited part of the alimentary canal, evidenced by a sore mouth, copious solid stools (without diarrhœa), and progressive emaciation. (Emaciation is rare, but anæmia may be very great—*Galloway.*)

5. *Intestinal Type.*—No ulceration of the mouth, and but little dyspepsia. Stools liquid, copious, pale, and frothy.

6. *Modified by Treatment.*—The sore mouth, dyspepsia, and diarrhœa may subside, but the stools remain copious, and the emaciation continues to progress.

Prognosis is good for recent cases under proper treatment. It is bad in the case of long-standing cases, aged people, malarious subjects, or refractory patients.

Morbid Anatomy and Pathology.—All organs are distinguished by *extreme anæmia* and general *atrophy*. All the

mucous membranes of the body—including uterine, vaginal, nasal, &c.—show a sclerosis (*Galloway*).

The tongue lacks its epithelium, and the papillæ are obliterated.

The *alimentary tract* contains the characteristic lesions. The bowel is enormously thinned. The serous coat is healthy, the muscular coat atrophied.

From mouth to anus the mucous lining is eroded superficially, and atrophied interstitially.

The villi and Lieberkuhn's follicles are atrophic, and the solitary glands and Peyer's patches have practically disappeared.

Here and there pin's head indurations with pigmented circumferences are met with, being dilatations of follicles filled with a grumous purulent material.

Fatty or cirrhotic degeneration may be observed in places.

The *mesentery* and *omentum* are atrophied, and the *mesenteric glands* fibrotic.

The *liver* is small, pale, and flabby; and occasionally cirrhotic.

The *pancreas*, *spleen*, and *kidneys* are usually atrophied, and may be very cirrhotic.

In view of the dark bilious diarrhœa which often ushers in the disease, *Manson* thinks that hyperactivity of the liver is probably the first step in its development, followed by an exhaustion of the hepatic chologenic functions.

Analyses of the stools have been undertaken at various times. *Wynter Blyth* found bile elements; *Bertrand* and *Fontan* failed to do so.

Micro-organisms, of course, abound, but none are pathognomonic.

During life, the primary lesion found by *Galloway* in all cases examined microscopically from sections of the tongue, proved to be a round-celled infiltration, not of the mucosa, but of the submucosa.

Treatment.—This is practically purely dietetic, very little dependence being placed on any drugs.

There are three chief types of dietetic treatment, each of which has its earnest partisans.

Milk is perhaps the earliest and most widely adopted treatment, but, as *Cantlie* points out, the return to solid stool (by patients on that treatment) merely means that the patient is living on whey, and passing a non-fæcal cheesy curd, which does not stimulate the functional activity of the liver, as seems to be demanded by its subnormal and atonic condition.

The following then are the three treatments:—

I. **The meat treatment** (*Cantlie*).—(*a*) The patient is put to bed.

(*b*) A hot wet pack is applied from nipples to groin. Kept on for two hours by a large bath towel. Repeated each night and morning.

(*c*) Unless extremely ill, 5 ozs. of pounded beef, lightly cooked, are given three times a day.

(*d*) Beef tea, beef jelly, calve's foot jelly or plain jelly are given every two hours during the day, and also at night if awake.

(*e*) Castor oil ($1\frac{1}{2}$ drachms) is given every morning for the first three days.

(*f*) Santonin gr. iij. morning and evening for three days.

(*g*) Strawberries 3 to 4 lbs. per diem between meals (first advocated by *Thin*).

This meat treatment should be carried out early and systematically. By the third day, in all probability, no stool is passed after the first preliminary copious and loose stools. By the fourth day the motion is probably fairly solid, fæcal, and bile-stained. If so, the diet may be increased, a poached egg added to the beef at breakfast and dinner; pounded chicken at mid-day instead of beef. On the sixth day the meat or chicken may be finely minced instead of pounded. By the eighth day the patient may have a cut off an undercut.

As soon as the stools are perfectly solid, add vegetables to the diet—stewed celery, stewed seakale, or vegetable marrow. Pulled or baked bread may then be given, and the diet gradually approximated to that of a normal person.

The liver rapidly attains its normal size and functions under this treatment, and all the symptoms of the disease quickly clear up.

II. The **milk treatment** (as recommended by *Manson*).—(*a*) A dose of castor-oil.

(*b*) The patient sent to bed, with flannel binder to the abdomen.

(*c*) After oil has acted commence milk treatment:—3 pints of milk per diem, given every hour by teaspoon or straw.

(*d*) When stools more solid, mouth less sore, and abdominal distention relieved, then increase quantity of milk by $\frac{1}{2}$ pint per day up to 100 ozs., and continue at this amount for ten days.

(*e*) Patient may now get up. Milk should be increased up to 7 pints per diem, and continued for six more weeks.

(*f*) A raw egg or digestible starches may then be given, and normal diet gradually attained by stages of fish and chicken.

(*g*) Strawberries allowed throughout.

III. The **fruit treatment** (as adopted in the home of sprue, Java).—Large quantities of fruit are given, either fresh or preserved, without sugar — strawberries, apricots, peaches, apples, pears, grapes, bananas, mangosteens, cucumbers, melons, pumpkins, and other watery fruits, such as the juice of oranges and pumeloes.

Pine apples and sour fruits are not allowed.

In all the above treatments, wines, spirits, coffee, &c., should be prohibited, and the use of tobacco forbidden. The consensus of opinion would seem to be that, in the most profoundly acute cases, the milk treatment is preferable at first to the meat treatment. If sprue develops in the tropics, the patient should be sent home as soon as possible, but not if diarrhœa is active, or the patient's end is near.

Great care should be taken during the voyage to avoid chills. Next to warm clothing, avoidance of fatigue is also essential.

The sprue patient should, if possible, not return to the tropics.

compelled to do so, he should take the greatest care of his health, and avoid exposure, fatigue, cold baths, alcohol, and all excesses.

Drugs.—Many drugs have been recommended, but, with the exception of those mentioned in the above treatments, they are of small avail, except in dealing with special symptoms as they arise.

Hartigan advocates *cyllin.* Palatinoids of 3 minims are given after food every two hours. The drug should be continued for a month after all symptoms disappear. *Galloway* recommends chinosol, gr. 5 thrice daily, and gradually reduced. If anæmia is marked, *Manson* uses intramuscular injections of minute doses of arseniate of iron.

Opium may be resorted to for severe abdominal pain or violent diarrhœa, but great care must be exercised in its administration.

Constipation occasionally demands the use of mild aperients, such as castor-oil, rhubarb, &c.

For great *flatulence,* powdered vegetable charcoal may be tried.

In *Shanghai* various mercurial preparations are in use; also the following tripartite treatment:—

1. *Rein's mixture* (cinnamon and simaruba).
2. Powdered cuttlefish bone (calc. carbonate) in large doses of 2 teaspoonfuls at a time.
3. Alteratives.

For *hill diarrhœa* Crombie recommends liq. hyd. perchlor., ♏ x., before each meal.

For the *buccal lesions.* The tongue may be pencilled with 2 per cent. cocaine solution before meals. A chlorate of potash gargle may be frequently advisable, and the ulcers touched with silver nitrate.

Cacao butter or other bland fat applied to the tongue, before eating a meal, is also an occasional relief to the patient.

Van der Burg recommends a gargle with a tincture made from the rind of *Pterocarpus indicus.*

During convalescence the careful use of tonics is indicated, such as wine, quinine, iron, and arsenic.

Relation to Hill-diarrhœa.—This condition, frequently found in some parts of India, seems to be analogous to the preliminary diarrhœa which so often ushers in a typical sprue.

Dyson and *Duncan* consider that the presence of mica in the water supply may be a causative factor, but, as *Maynard* has shown, in Darjeeling mica has been practically banished from the water supply, yet hill-diarrhœa is still often met with. Moreover, in that district, the usual water drinkers (women and children) rarely suffer from the disease.

The probability is, that men arrive from the hot plains with liver functions impaired by the heat and bad food. Hearty eating and violent exercise, combined with indiscretions in clothing, aggravate their condition; the liver becomes hyperæmic; diarrhœa follows; and, if the patient's health has been seriously undermined in the plains, the diarrhœa continues, and typical sprue may develop.

CHAPTER XXXV.

TRYPANOSOMIASIS.

Definition.—A protozoal infection of human beings by *T. gambiense*, conveyed from sick to healthy by the bite of a tsetse fly, *Glossina palpalis* (possibly also by other means); and characterised by chronic irregular fever, patchy erythema, anæmia, and enlargement of lymphatic glands.

It terminates either in recovery or, more often, in death—death being due to (*a*) an aggravation of the original infection known by the name of *sleeping sickness;* or (*b*) intercurrent affections, such as pneumonia.

Synonyms.—The recognition of the condition being of recent date (*1902*) there has been no conjuring with names, such as invariably must take place in the nomenclature of these diseases whose specific etiology remains for centuries undiscovered.

Its late and serious phase, of which the clinical features have long been known as *sleeping sickness* has also been called *Negro lethargy; Schlafkrankheit der Neger; Maladie du Sommeil; Congo sickness; N'tansi* (on the Congo).

History and Geographical Distribution.—In the early part of the nineteenth century a brief account of sleeping sickness was published by *Winterbottom*. Other clinical investigators who have published accounts of this disease are :—*Corre* in Senegambia; *Clarke* (1840) in Sierra Leone; *Nicolas* and *Guerin* in the West Indies amongst imported slaves. Years went on and the etiology still remained a mystery. About 1890 a French observer *Nepveu* found a blood parasite of man which was probably a trypanosome.

In May, 1901, *Forde* of the Gambia Colony, in West Africa, had an English patient suffering from chronic irregular fever supposed to be malarial, but which failed to yield to quinine. Blood investigation demonstrated a minute non-filarial, worm-like, extra corpuscular organism.

After six months furlough in England the patient returned to the Gambia having frequent relapses of his old fever. *Dutton* who was then working at malaria in West Africa, being consulted, recognised the parasite as a trypanosome. This was the first occasion on which it was definitely recognised that man was liable to trypanosome infection. **Dutton** named the parasite **Trypanosoma gambiense**, and the disease **Trypanosomiasis**.

In October, 1902, *Manson* was consulted in London by a lady who had lived for some time on the Upper Congo, for chronic fever, supposed to be malarial. For over a year she had had a series of

febrile attacks (traced to the bite of an insect in August, 1901), owing to certain similarities of the clinical features to those of Forde's case Manson suspected trypanosomiasis. The patient was sent into hospital and many blood examinations gave negative results, until after a week *Daniels* found a solitary trypanosome. Subsequently, although occasionally none were found yet, generally, two or three were present in every film and sometimes as many as seven or eight. The lady's symptoms persisted until in October, 1903, she became bedridden. Symptoms resembling "sleeping sickness" developed and ran a rapid course, death taking place in November.

Mott in an histological examination found the small-celled perivascular infiltration of brain and other tissues characteristic of morbid pathology of sleeping sickness.

This case as well as other facts are almost conclusive of a trypanosome origin of sleeping sickness.

Soon afterwards *Dutton* found the parasite in the blood of a native Gambian child, but with no concurrent symptoms.

Manson, recalling the case of a lady missionary patient from the Congo whom he had treated for much the same symptoms some years before, wrote to Africa for information, and was told that *Broden* had found the trypanosome in her blood.

In June, 1902, owing to the rapid progress and ravages of sleeping sickness in Uganda, the British Government sent out a Royal Society Commission to investigate the etiology and possible connection with *Filaria perstans* which had very frequently been found in the blood of people suffering from the disease. One of these Commissioners—*Castellani*—found a trypanosome in the centrifugalised cerebro-spinal fluid of one, and subsequently in others of his cases, as well as in the blood. *Bruce* confirmed and extended this discovery; and we now know that the *T. gambiense* which causes the trypanosome fever on the West Coast, has been found in the blood, the cerebro-spinal and other serous fluids, and in the lymphatic glands of practically every case of sleeping sickness which has been competently observed.

As regards *the distribution of this disease* it was first recognised as endemic on the West Coast of Africa from Senegambia to Benguela, occurring in negroes, especially a little back from the Coast. Every now and then in endemic areas it would become epidemic.

It the old days of slavery amongst the Africans in the West Indies, sleeping sickness would break out months or even years after having left the West Coast, but was absolutely confined to those negroes who had come from Africa and never spread amongst the others.

Apparently in consequence of increased commercial facilities and routes, the disease extended its endemic area from the upper Congo basin in the north to the Portuguese Colonies in the south until many of the riverside villages throughout the Congo Free State were affected. About a decade ago (in the latter part of the nineteenth century) it appeared on the east side of the Continent, and spread from

the upper part of the Nile Valley, invading Uganda and all the region to the north of Victoria Nyanza, where some 40,000 are estimated to have died from it within a few years.

Etiology.—Perhaps the disease in question, more than any other, will show the danger of jumping to too hasty conclusions. The theories have been legion :—

An intoxication by moulds of maize, rice, or other cereals; palm wine, Indian hemp, nostalgia, sunstroke, scrofula, malaria, beri-beri, embryos of *Rhabdonema strongyloïdes*, ankylostomiasis.

Then came a bacterial theory. Two members of the Portuguese Commission—*Cagigal* and *Lepierre*—discovered a micrococcus, with thickened extremities associated with inflammatory exudate, in the brain and meninges. They cultivated it, and are said to have induced a similar disease in rabbits by injections.

Before he found the trypanosome, the British Commissioner, *Castellani*, found a similar micrococcus in a large proportion of the cases, till he, too, at one time thought it possibly might be the specific agent.

Manson suggested that *Filaria perstans* (which he had himself discovered in 1891) was very possibly the specific agent, and this on extremely plausible grounds. It was found in the blood of a negro who died from sleeping sickness in a London hospital. Two more cases brought to London showed large numbers of this filaria in their blood; moreover, the geographical range of the disease and parasite apparently coincided. Ninety per cent. of cases of sleeping sickness at Entebbe, in Uganda, proved to have *F. perstans;* in fact, the argument in its favour was a very strong one. On extending the enquiry, however, it was proved that a large proportion of natives in the endemic area harboured *F. perstans*, but showed no sign of sleeping sickness; in other districts sleeping sickness was prevalent, but *F. perstans* absent. In Demerara 60 per cent. of the Indians suffered from filarial infection, but sleeping sickness was not known. It was obvious, therefore, that the association of the two was merely a matter of coincidence.

Then came the finding of *Trypanosoma gambiense* in the blood of a fever patient, and its subsequent discovery in cases of sleeping sickness, as has been dwelt on already in this chapter.

The following **tabular summary of modern etiological knowledge** is compiled from *Bruce*, *Novy* and *M'Neal*, *Koch*, *Greig*, *Gray*, and other sources :—

1. The trypanosome found in human blood in W. Africa, Congo, and Uganda is identical with that found in cases of sleeping sickness, as proved both by morphological similarity and by the identical results which follow animal inoculation.

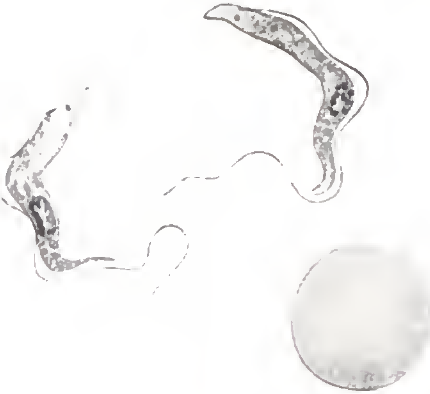
2. This trypanosome is Dutton's *T. gambiense.*

3. Trypanosome fever is only the first stage of sleeping sickness.

Europeans and natives with trypanosomes in the blood have, after the lapse of a year or more, developed fatal and typical symptoms.

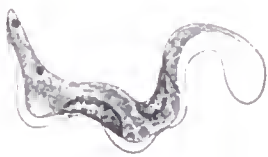
4. The incubation period may last for years.

5. After infection, death will eventually occur from the disease in the very large majority of cases.

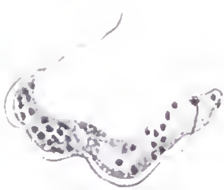


T. gambiense from the blood of a Gambian native.

T. gambiense from the blood of a tame rat.



T. gambiense. Longitudinal division, showing two nuclei and two blepharoplasts.



T. gambiense. "Stumpy form."

T. gambiense. "Round form."

Fig. 74 (*after Dutton*).

6. By whatever other channels the disease may be conveyed from e sick to the healthy, it has been proved that a particular tsetse —the *Glossina palpalis*—acts as an intermediate host.

7. There is an identical distribution of this fly and sleeping kness; where the fly is absent there is no sleeping sickness.

S. The fly has been found in Africa, from 28° S. to 15° N.

9. There is a certain amount of evidence to show that other ecies of tsetse fly can also transmit the disease.

A diseased unit, therefore, if introduced into the habitat of such would prove a grave source of danger.

110. Other biting flies, such as the horse fly (*Tabanus*) or stable (*Stomoxys*), apparently cannot transmit the disease.

111. The endogenous cycle of *T. gambiense* in the human host is mplete, the organism dividing by longitudinal division.

112. There is an exogenous cycle in *Glossina*. They multiply pidly by longitudinal division, increase in size, and show differen-tion in form (sexual types). Some increase in thickness; exhibit undant protoplasm, taking a blue stain; and possess a loosely ticulated, large, round, chromatin body (female).

Others have a very slender appearance, possess a long slender romatin body, and have no blue-staining protoplasm (male).

The fertilised female is probably represented by the stout forms th multiple nuclei, which are often found.

Simple, globular, nucleated cells—exhibiting transitional phases the typical mature structure—are often found and are probably ughter cells split off from the fertilised female, corresponding to e number of the latter's nuclei.

113. A micrococcus is fairly constantly found, *post-mortem*, to be sociated with sleeping sickness. This only invades the system at e very last stages of the disease.

114. The trypanosomes have been cultivated by *Novy* and *M'Neal*, *vitro*, on a mixture of agar and blood.

To ordinary agar (cooled to 45°) is added defibrinated aseptic ood, and the mixture cooled in flasks giving as large a surface yer of condensation water as possible.

Blood to agar: 2 to 1. The incubation temperature should be ° C.

As soon as the growth is established, subcultures can be made.

Asepsis is essential. Cultures passed through a Berkefeld filter e a still infective filtrate.

15. The leucocytic variation in trypanosomiasis resembles that malaria. There is a constant relative increase in the large ononuclear elements, which constitute 20 per cent. or more of the icocytes found.

Species of Tsetse Flies (*Austen*).

1. *Glossina palpalis.*—Uganda, Sierra Leone, Northern Nigeria, d Congo Free State. Is a river fly. No connection with big

game. ? Wild pig. Pupa, 5 to 6 mm. long, 3 mm. broad. Conveys trypanosomiasis.

2. *G. pallicera.*—Assinie (W. Africa).

3. *G. morsitans.*—Zululand, Transvaal, Portuguese East Africa, Rhodesia, British Central and E. Africa, Togoland, Nigeria. Transmits fly disease of domestic animals.

4. *G. tachinoides.*—Northern Nigeria, Chad, and Shari River.

5. *G. pallidipes.*—British Central and E. Africa, Uganda.

6. *G. longipalpis.*—Togo, Zambesi region.

7. *G. fusca.*—British E. Africa, Congo, Gold Coast, Ivory Coast, Togo, Nigeria, Mashonaland. Bites at night sometimes. Conveys *Ngana.*

8. *G. longipennis.*—British E. Africa, Somaliland.

Trypanosomata of Mammals.

1. *T. lewisii.*—24 to 25 $\mu \times 1$ to 4 μ. Found in a small proportion of sewer rats in England. Is non-pathogenic. Common in tropical rats. More slender than *T. brucei.* Stated to be conveyed by rat fleas.

2. *T. brucei.*—25 to 30 $\mu \times 1 \cdot 5$ to $2 \cdot 5$ μ. Is the highly pathogenic trypanosome of *Ngana* (or tsetse fly disease). Fatal to nearly all mammals. In the horse produces:—Watery discharge from eyes and nose, abdominal œdema, anæmia, and wasting. Is conveyed by bite of *G. morsitans.*

3. *T. evansi.*—20 to 30 $\mu \times 1$ to 2 μ. Is the trypanosome of *Surra*, a disease of cattle common in India. Symptoms similar to those of ngana. Immunity against ngana does not confer immunity against surra. Disease said to be conveyed by horse flies (*Tabanidæ*).

4. *T. equinum.*—20 to 25 $\mu \times 2$ to 3 μ. Is the trypanosome of *Mal de Caderas.* Is endemic in Central and South America. Is a chronic disease of horses and donkeys. Symptoms similar to ngana and surra. There is also paralysis of the hind legs. The course is chronic (two to twelve months). Incubation period, five to eight days. Occasionally hæmoglobinuria. Rats, rabbits, dogs, guinea-pigs, &c., can be infected. Infection carried by a biting fly (*Stomoxys calcitrans*).

5. *T. equiperdum.*—18 to 26 $\mu \times 2$ to $2 \cdot 5$ μ. Is the trypanosome of *Dourine.* A disease of horses in Algeria and India. Incubation period, eleven to twenty days. Infection conveyed by coitus. In forty to fifty days, after previous œdema of genitals, characteristic plaques occur on the skin, and last for one to eight days. Animals become anæmic, paraplegic, and die in two to ten months.

6. *T. gambiense.*—18 to 25 $\mu \times 2$ to $2 \cdot 8$ μ. Found by Dutton at the Gambia. Is first human trypanosome found. Occurs in two main forms:—(1) A long form with pointed posterior end; and (2) a short stumpy form with many chromatic granules. It is pathogenic for many animals—*e.g.*, rats, guinea-pigs, rabbits, and monkeys (except *Cynocephalus*). Disease is a chronic one in animals. Recovery may take place.

7. *T. theileri.*—30 to 65 $\mu \times 2$ to 4 μ. Is the trypanosome of *Galziekte*, a South African disease of cattle. Is transmitted by a biting fly (*Hippobosca rufipes*).

8. *T. transvaliense.*—18 to 50 $\mu \times 4$ to 6 μ. Is a South African cattle trypanosome. The centrosome almost touches the nucleus, consequently the undulating membrane is but little developed.

Various other trypanosomes have been described in cattle, horses, or camels in Uganda, Algeria, Soudan, Somaliland, Togoland, Annam, Mauritius, the Philippines, Java, India, &c. The largest, so far discovered, was by *Falshaw* in Singapore (1906), who found a trypanosome 75 μ in length in the peripheral blood of a bullock. The bullock died in three days, but no further parasites were discovered, either in the spleen or cerebro-spinal fluid—nor could any macroscopic morbid process be found.

Symptoms.—A probable history of an insect bite will be made out, with a painful swelling at the spot, which slowly subsides.

After an incubation period of about a fortnight, a febrile attack comes on, the temperature rising to 100°, 101° F. or more, and generally dropping to normal each morning. Palpitation is complained of, and breathlessness; and there is a progressive muscular weakness. These attacks come and go with intervals of complete apyrexia. Quinine has no effect whatever on the course of the fever.

The spleen gets gradually enlarged; there is a puffiness of the face; and a patchy erythema is generally to be seen, irregularly distributed over the limbs and trunk. One or more glands will become enlarged—femoral, inguinal, or superficial cervical.

Anæmia becomes marked, and the pyrexial attacks continue.

Eventually the disease will terminate in one of three ways:—

1. *Recovery.* This is rare.

2. *Intercurrent disease*, such as pneumonia, will cause the patient's death; or

3. *Sleeping sickness* will supervene.

The symptoms of a chronic meningo-encephalitis will gradually exhibit themselves. The low febrile disturbance continues, accompanied by headache. Lassitude is established, gradually becoming more marked, until there is profound physical and mental lethargy. The patient becomes bedridden; muscular tremors are observed, chiefly of the sterno-mastoids and limb flexors. Paresis is common; there is incontinence of urine. With the exception of some little amnesia, the mental functions remain intact. The superficial reflexes are, as a rule, normal.

The alimentary functions gradually decline; bedsores develop; the somnolence gives way to a deep stupor, and death by convulsions or exhaustion takes place.

Pathological Anatomy.—The following are the usual *Macroscopic appearances*:—

1. General glandular enlargement.

2. Increase of subarachnoid fluid, with flattening of the convolutions.

3. Dilated and dropsical brain ventricles.

4. Congested points in the brain substance.

5. Minute hæmorrhagic areas in the stomach (not constant).

Microscopic appearances are:—

1. Peri-vascular small-celled infiltration in brain and spinal cord—especially noticeable for the presence of plasma cells.

2. Hæmorrhagic lymph glands.
3. Necrotic areas in the spleen.
4. Degenerated bone-matter.

Treatment.—Investigators, such as *Bruce*, *Laveran*, *Thomas* and *Breinl*, have noted the marked action of arsenic on trypanosomes. The protective effect, however, proved to be merely temporary, and after a little the parasites re-appeared in spite of increasing doses.

Thomas and *Breinl* thought it might be feasible to get an arsenate with an organic base which would produce the same effect on the trypanosomes, but prove less toxic to their host.

I. *Atoxyl* was, therefore, tried. The formula is $C_6H_5NHAsO_2$ (meta-arsenic-anilid), containing about 37·6 per cent. of arsenic.

Large quantities have been sent to W. Africa, and the results are so far very satisfactory.

Broeden, *Daniels*, *van Campenhout* and others report very well on it.

It is a mistake to give the drug by the mouth, as it is broken up by the acid contents of the stomach, and toxic effects are more easily produced.

The drug should be administered intramuscularly in 5 to 10 per cent. solution. Begin with a dose of 0·2 gram of atoxyl, increasing daily by 0·05 gram till a dose of 0·8 gram is reached. If there are no toxic symptoms, the dose should remain at this for a fortnight or three weeks; then gradually lessened by 0·05 gram daily until a dose of 0·2 gram is reached, when the treatment may be interrupted for one or two months. Three or more such courses are given as required. *Kopke* of Lisbon does not report so favourably on his results with this drug (1905-7).

The *toxic symptoms of arsenic* are:—Vomiting, diarrhœa, burning on micturition, dryness of the skin, pruriginous vesicles, slow pulse, cold extremities, and pectoral cramps.

Other forms of treatment are:—

II. *Trypan-red*, an aniline dye, of which the dose is 5 grains; and

III. *Chrysoidin*, which *Neave* has used with success.

The dose is $\frac{3}{20}$ grain used hypodermically, and gradually increased up to $\frac{1}{2}$ grain.

Mense (1905) has suggested X-rays in the treatment of trypanosomiasis.

Ross, however, experimented by submitting living trypanosomes for periods of half to one hour to very strong rays of three kinds—Röntgen, Finsen, and those emitted by radium. No appreciable effect on the organism was observed.

CHAPTER XXXVI.

VARIOUS MINOR DISEASES.

GLANDULA IDIOPATHICA.

nonyms.—*Climatic bubo; non-venereal bubo.*

History and Geographical Distribution.—From many ts of the world cases of glandular enlargement (chiefly inguinal) re been reported, where none of the usual causes could be found, h as sexual diseases, skin diseases, injuries, plague, or filariasis.

Ruge, in 1896, reported 38 cases amongst the blockading adron on the Zanzibar coast in 1888-89.

The affection is often observed in the Navy. In the East Indies . China stations, between 1884-94, with a mean strength of 180 men, an average of 733 cases were observed annually. On er stations the reports have been as follows:—West Indies, 22 1,000; West Coast, 13 per 1,000; Channel and Home Fleets, per 1,000; Australia, 9 per 1,000; and the Mediterranean, per 1,000.

Skinner notes 49 cases that occurred in a regiment and one tery, of which 28 were in Calcutta, 13 in Hongkong, 4 in gland, 2 in Allahabad, and 2 in Malta.

Vagel has reported many cases in German East Africa.

Scheube noted 16 cases in Japan.

Martin observed many cases at Deli, in Sumatra, chiefly amongst malarial cachectic patients.

Lesueur - Florent saw several cases in Madagascar, of which occurred on an insalubrious ship.

The author has had one case from a British man-of-war under eful observation in Singapore.

Etiology.—The etiology of the disease is highly obscure.

Most of the recorded cases have been in young men of 17 to 30 rs of age.

No special season of the year seems to be associated with its urrence, though the majority of Ruge's and Scheube's cases urred in the spring and autumn.

Practically all observers deny a connection with malaria, except *rtin*, who does not state if any blood examinations were made in cases. In all cases where blood examinations have been made, parasites have been found.

A venereal origin has been asserted by some, but the careful enquiries pursued in so many of the cases, and the strict observation to which naval cases are subjected, should certainly preclude any liability to such a diagnostic error.

Cantlie identifies the cases with pestis minor. Cases of pestis minor or plague buboes, unaccompanied by grave constitutional disturbance, are doubtless common in Hongkong, as they are in all plague centres, and possibly for this reason Cantlie may doubt the separate existence of Glandulæ idiopathicæ; but the fact of the occurrence of the latter in naval stations at home and in the West Indies (where plague has not been reported) and amongst the class in which plague would seldom get a footing, should enable us to exclude such an etiology, especially when those cases which have been examined have given no sign of the plague bacillus.

Scheube considers that climate has undoubtedly something to do with it. The fact that cases occur not only in the tropics, but under such different conditions as the temperate climates of England and Japan, should be sufficient to convince us that some other factor must be at work.

Symptoms.—There may be a few prodromal symptoms, such as malaise and headache, but the disease is usually ushered in by fever. In a few cases there is an entirely afebrile course.

At or about the occurrence of the fever begins an enlargement of one of the lymphatic glands. This is usually a unilateral inguinal lymphadenitis; more rarely it may be bilateral, or the femoral glands may be affected. The enlargement develops quickly, and may be of any size from that of a sparrow's to that of a goose's egg. The swelling is indurated, and usually painful on pressure. About 60 per cent. of these glands become eventually absorbed; the remaining 40 per cent. will go on to suppuration.

The type of fever is an irregularly remittent one. The usual course of the disease is about three weeks, but it may be indefinitely prolonged.

The accompanying chart is of a case under the author's care. An A.B. of a British cruiser was reported as sick on the arrival of the vessel, having suffered from four days' fever and a right inguinal bubo. The man had been in England for some time. The only ports of call were Malta and Colombo, at which latter place the man was ashore for a few hours. Six days after leaving Colombo the man was reported sick with fever and an indurated bubo. The day after admission the bubo was incised with a view to lessening tension.

On arrival at Singapore the possibility of a pestis minor was considered. A microscopical examination of the bubo smear, however, gave a negative result; nor were malarial parasites found in his blood. There was no history of malaria. His spleen, lungs, and liver were normal.

A venereal origin was entirely excluded. His subsequent history can be seen from the chart (Fig. 75). The diurnal intermittence of temperature during the second week suggested a possibility of tubercular mischief, but a Ziehl-Neelsen examination of a bubo section gave

a negative result. The induration began to diminish at the end of the second week, and the glandular swelling at the end of the third week. At the end of the fourth week he was discharged cured.

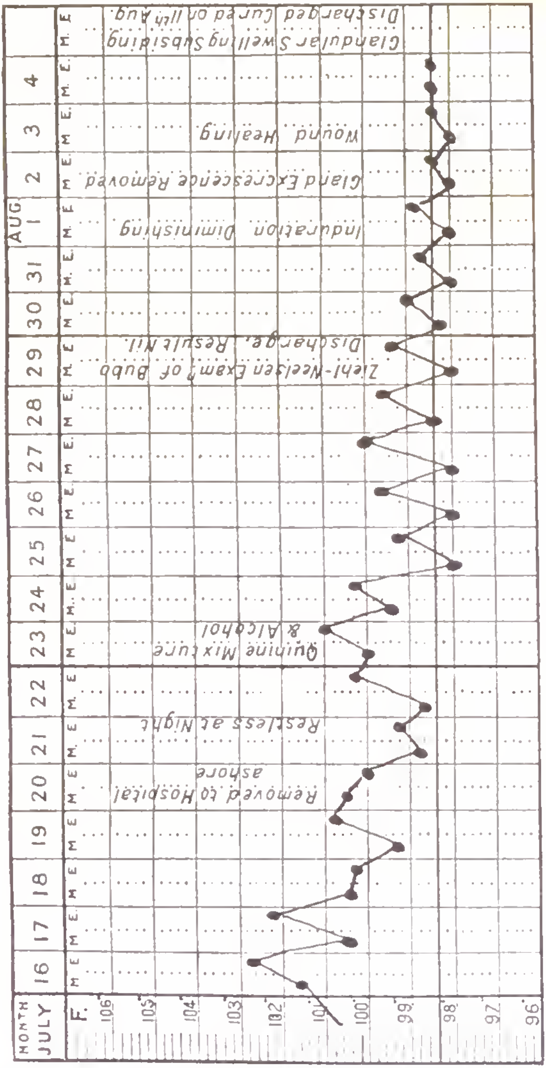


Fig. 75.—Temperature chart. Glandula idiopathica.

The uncomplicated mortality is *nil*. The morbid anatomy and pathology have, therefore, not been properly worked out.

The complications and sequelæ are few. Sleeplessness may be considerable. The urine will, in some cases, show a trace of albumen.

Treatment.—This is merely a matter for rational procedure on general lines.

At the commencement a smart purge will be beneficial.

If malaria has been excluded, quinine will prove useless.

The bubo should be painted with iodine, and should be promptly opened if suppuration occurs.

The following prescription should be tried, and will probably prove beneficial :—

℞.—Calc. iodidi, gr. ij., t.d.s.

The bowels should be kept open by a seidlitz powder when necessary.

Sleeplessness may demand a dose of bromide.

If the patient has been used to alcohol it should not be withheld, but can be given with advantage throughout.

A tonic is indicated during convalescence.

AINHUM.

A progressive constriction of one or more toes.

Synonyms.—*Ringtoe*, *Banko-kérendé* (in the Soudan), *Sukha-pakla* (in India).

Distribution.—The disease occurs chiefly in negroes. It was first reported by *Clarke* on the west coast of Africa in 1860. Mention is also made of it in Brazil, North Africa, British Guiana, Madagascar, and some of the Pacific Islands.

It is occasionally seen in Hindus in India. *De Brun* reports two Semitic cases in Syria, and *Freer* has seen a doubtful case in Penang in an adult Chinese male.

Symptoms.—The disease commences with a superficial thickening of the digito-plantar fold of one little toe, or more rarely of both little toes.

Da Silva collected five cases in which the fourth toe was affected. *Gongora* has noticed it once on the second toe, and *Cooper* once on the big toe. *De Brun* states that in one case all five toes of one foot were attacked.

The superficial thickening gradually becomes constricted, forming a groove which, in the course of several years, completely surrounds the toe.

The groove deepens, and the fibrous base of it gradually promotes automatic amputation.

Ulceration may or may not be present either at an early or a late stage. Its presence would appear to be more or less adventitious.

There is seldom any pain or hindrance to locomotion.

If the disease progresses unusually fast, there may be some con-

siderable pain from pressure effects on the nerve. An ulcerated condition will also frequently prove extremely painful.

Etiology.—While the exciting cause is not definitely known, there can be little doubt that heredity has much to do with the disease.

Da Silva and *Manson* think that wounds from sharp grasses, &c., by keeping up continual irritation whilst walking barefoot have much to do with it. This is doubtless in many cases an exciting cause. The author, who came across many cases amongst the negroes of the Bahamas, generally found a family history. In some cases, great-grandfather, grandfather, father, son, and grandson had all been affected. Very seldom more than one in each generation seemed to be attacked, though occasionally the disease would appear in the offspring of a patient's unaffected brother.

Women seem to be very seldom afflicted with the malady. As a rule, it does not develop even in males until after puberty.

Pathological Anatomy.—As the strangulation advances, it first causes a slight œdema. This is followed by sensory changes, and the tactile sense is gradually lost. Hyperplasia of the subcutaneous adipose tissue of the affected toe is always found, and may be excessive. The bone of the toe is partly absorbed, and is infiltrated with fatty cells.

The constricting ring is of fibrous tissue. *Manson* reports a caudal affection in a pet monkey of a somewhat similar nature.

Treatment.—As a prophylactic measure special care of the feet should be taken in all cases with a family history by hardening the skin and avoiding any sources of local irritation.

Division of the constricting fibrous band at an early stage of the disease has been suggested as a means of delaying the evolution.

Failing this, amputation is the only treatment.

GOUNDOU.

This is a disease affecting the nasal processes of the superior maxillæ which occurs amongst negroes, and has a West African distribution.

The disease usually commences soon after childhood, or more rarely during adult life.

A persistent frontal headache accompanied by a sanguino-purulent nasal discharge precedes or ushers in the formation of symmetrical osteomata of the nasal processes.

The other symptoms subside in a few months, but the bony tumours increase even up to the size of an orange, and may encroach on the eyes.

Pathologically the tumours consist of thin compact bone with a cancellous core. The skin over them is normal and movable.

Etiology.—Nothing is known as to the cause of the condition.

Chalmers considers them the product of an osteoplastic periostitis, and thinks they may have some connection with yaws.

Macland has suggested dipterous larvæ in the anterior nares as an exciting cause.

The affection is not hereditary, and is more common in men than in women. It is supposed also to affect monkeys.

The removal of the tumour is the only effective treatment.

LATAH.

A species of cerebral neurosis occurring chiefly in the Malay Archipelago and Peninsula, and in Burma and Siam, characterised by involuntary movements or sounds induced by fright or by suggestion.

The imitative and impulsive symptoms may arise from fright, or may at any time be brought on by demonstrative movements in front of such persons, which are promptly mimicked (echocinesia), and words spoken are repeated (echolalia); orders given are also promptly executed.

A look, a touch, a motion of the face or head are often enough to start a *séance*.

The exclamations and movements of the Latah are quite involuntary, and cannot be restrained by the most strenuous exertions.

It does not resemble the hypnotic state in that consciousness is maintained, and the intellect is undisturbed.

As a rule, other nervous disorders are not present. *Van Brero*, who has made a considerable study of the disease in Java, where it is very general, has never been able to confirm hysteria or epilepsy in his patients.

The disease occurs chiefly in *women*, especially young ones.

It appears to be incurable.

Van Brero connects the weakness of will power with the defective development of character of the Malays, due no doubt to neurotic temperament. He considers that heredity plays an important part in the disease.

The author has investigated several cases in Singapore—all occurring in men. In these no family history could be obtained. Excessive venery was an acknowledged feature, and may have something to do with the etiology. At least it is worth investigation, for the Malays are often credited with an insatiable sexual appetite.

The following are the author's notes of one Malay case which are perhaps worth recording.

Ahmat, ætat 45. Born in Singapore. Father born at Malacca, mother at Siak in Sumatra—both now dead. None of his relations ever had latah. No history of syphilis in parents. Had four brothers and two sisters. Two of former died of phthisis.

Personal history, &c.—Has been at sea for thirty years. Was first married at 12 years old. Has had eight wives, of whom six are dead. No history of epilepsy or syphilis, and has had no mental disorders. Had ague some years ago in Java.

The lungs show slight emphysema.

The heart is rather hypertrophied.

No disturbance of cutaneous sensation. Has had shooting (stabbing) pains over legs and head for past six years.

The pupils react to light and accommodation.

The knee-jerks are both absent.

He cannot walk a straight line, and is somewhat unsteady with eyes shut. KCSN is absent from the saliva.

Has had latah for ten years.

A sudden fright or loud noise will start an attack, during which he will mimic actions and words, and execute any orders, however foolish. After such a *séance* he is very tired. There is a history of occasional headaches.

CHAPTER XXXVII.

YAWS.

Frambœsia.

Definition.—A contagious, chronic, infectious disease of warm climates, characterised by an eruption, at first fungating and afterwards encrusted; and which tends slowly to spontaneous cure.

Synonyms.—*Frambœsia* (Sauvages, 1759); *Pian*, in Martinique, &c.; *Coco*, in Fiji; *Parangi*, in Ceylon; *Gattu*, West Coast.

History and Geographical Distribution.—In 1525 *Oviedo* reported the disease by the Spanish name of "bubas," having observed it in San Domingo.

During the seventeenth century *Piso*, *Bontius*, and *Labat* wrote accounts of it from Brazil, the West Indies, &c. Some years ago the subject was largely elucidated by the labours of *Milroy*, *Bowerbank*, *Kynsey*, and others, while recently *Numa Rat*, *Nicholls*, and *Castellani* have further extended our knowledge of the subject.

The early endemic home of the disease was probably situated in West Africa, and from there was carried to the West Indies in slavery days, and spread gradually to some of the South American republics.

In Asia the distribution is throughout the Malay Peninsula and Archipelago, with the countries to north of them—Assam, Burmah, and Siam.

It is also found on parts of the Indian coast, in Ceylon, and in some parts of China.

There is an endemic focus in several of the Pacific Islands, of which Fiji is a noted example.

Etiology.—There is an *incubation period* of twelve to twenty days, as proved by the inoculation experiments of *Paulet* and *Charlouis*. Some authors give two months as the incubation period of the acquired disease, probably owing to errors in determination of the date of the infection.

One attack confers immunity.

Either sex is equally affected, and no age is exempt, but two-thirds of the cases occur before puberty.

No race is exempt, although black and coloured people are more frequently attacked.

Yaws is highly contagious, but only when the contagium is

introduced through abrasions of the skin. In addition to this direct contact, it is possible that some biting insects may occasionally act as intermediate hosts.

Yaws is neither congenital nor hereditary; nor is it conveyed by suckling.

The disease has been thought by many to be a manifestation of syphilis modified by race and climate.

This was first suggested owing to the similarity of some of the local lesions occurring in the two diseases, and some support has been lent to the view by the finding of certain spirochætes by *Castellani* in cases of yaws, bearing a great resemblance to the *S. pallida* of syphilis.

That these diseases are separate entities, however, can scarcely be doubted. *Charlouis*, in 1881, successfully inoculated a yaws patient with syphilis. *Powell* reported two cases of yaws in men who contracted syphilis while still suffering from the former disease. More recently, *Neisser's* and *Baermann's* experiments with monkeys in Java demonstrated the clinical differences, and showed that syphilis and yaws do not influence each other in their development in monkeys.

The following table will show at a glance the chief differences between the two diseases:—

Yaws.	Syphilis.
Tropical distribution.	World-wide distribution.
Not hereditary or congenital.	May be both.
Not contracted, as a rule, by sexual intercourse.	Often so contracted.
Over 50 per cent. of cases occur between the ages of two and ten years.	Majority of cases occur in adults.
Primary lesion is soft ulcer.	Primary lesion is a hard chancre.
There is much itching.	There is no itching.
Hair never falls out.	Hair falls out in secondary stage.
Secondary eruption appears after six weeks.	
Secondary eruption asymmetrical.	Eruption more or less symmetrical.
Secondary eruption monomorphous.	Polymorphous.
Secondary eruption fungating.	No fungoid growth.
No lesions of mucous membrane of fauces.	Common.
No eye affections produced.	Common.
Neither viscera nor nervous system is attacked.	Common.
Bones only attacked from without by inflammatory extension.	Bones attacked from within
Great extravasation of polynuclear leucocytes.	Extravasation to less extent.
Hyperkeratosis marked.	Not marked.
Proliferative changes in epithelium very pronounced.	Less so, except in case of condylomata.
Infiltration of plasma cells only slight.	Infiltration is denser.

Yaws.	Syphilis.
Giant cells and chorisplaques never observed.	Frequent.
Collagen slightly resistant and never organised.	More resistant, and organised.
No transitional branching connective tissue cells.	Present.
No marked thickening or proliferation of the vascular endothelium.	Usual.

Frequent and long search has been made for the specific cause of the disease.

Pierez in 1890 first reported micrococci in yaws tubercles, which he was able to cultivate.

Nicholls and *Watts* in 1893 also found and cultivated micrococci. The same coccus was found in the organs of a case at post-mortem, and was also isolated from the dust of infected rooms; but animal inoculation yielded negative results.

Breda in 1895 found bacilli in tissue section in blood-vessels, but not intra-cellular.

Powell in 1896 found a blastomycetic infection in the granuloma.

In none of the above cases was the specific factor satisfactorily demonstrated.

In February, 1905, however, *Castellani* in Ceylon discovered some spirochætes in smears from ulcers of yaws.

Some of the films from Castellani's cases were sent by him to *Schaudinn*, who found therein three kinds of spirochætes, one of them closely resembling *S. pallida* of syphilis.

It is highly probable, therefore, **that yaws is really a spirochæte infection.**

The spirochætes found are as follows:—

I. In Ulcerated Lesions—(*a*) *S. refringens* (Schaudinn).—Is thick, and takes stain easily.

(*b*) *S. tenuis obtusa* (Castellani).—Thin, delicate, with blunt extremities, and varying number of spirals.

(*c*) *S. tenuis acuminata* (Castellani).—Is thin and delicate, but tapers at both ends.

II. In Non-ulcerated Lesions—*S. pallidula* (Castellani).—*Extremely* delicate and thin; tapers at both ends. Length up to 18 or 20 μ. Spirals numerous, uniform, and small. Morphologically is almost identical with *S. pallida* (Schaudinn).

Technique for Demonstration of the Spirochætes.—1. Make films in usual way, but useless after secondary pyogenic infection.

2. Apply Leishman for five minutes without previous fixing.
3. Add distilled water, and allow to act for several hours.
4. Wash with distilled water, leaving a few drops on the slide for a minute.
5. Dry and examine with highest power.

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Fig. 70.—Yaws.

(*After St. George Gray, Sierra Leone.*)

Symptoms (Fig. 76).—The inoculation experiments of *Charlouis* owed an ulcer at the point of inoculation in every successful case.

In the acquired disease, then, there is an infection through some ght cut or skin abrasion. If this infection be a slight one, then tle irritation may result, and healing occurs. Otherwise the wound comes inflamed and covered with a brownish scab, which conceals depressed and exuding sore. This ulcer may heal up before the neral eruption occurs (usually from five to seven weeks after the iginal infection); but, if large, it does not as a rule do so.

For a week or more before the general eruption, there is often a ght constitutional disturbance, manifested by transient attacks of rexia, headaches, indigestion, vague muscular pains, dry skin, and rhaps some enlargement and tenderness of the lymphatic glands. ese disturbances are usually more marked in children than in ults.

The general eruption may be widely scattered over the body, or ay be confined to certain skin regions. It consists of *papules*, at st small, but subsequently attaining the size of a pea. They are mmonest on exposed parts, and are most frequently found on the wer extremities.

The papules may remain discrete or become confluent.

The skin covering them gradually becomes thinner, and then oded, leaving rounded excrescences with a small, central, cheesy re. A yellowish serous fluid exudes from the surface, which dries form a yellowish crust.

These yaw-lesions are attended with much itching, but the yaw self is not sensitive, and lemon-juice or other acid may be applied ith impunity—a diagnostic point.

Within two or more weeks the maximum development of the crescences is attained.

The yaws then either (1) remain stationary for a further few weeks efore shrinking, disappearing, and leaving darkly pigmented areas; (2) instead of being absorbed, break down and ulcerate. These ay become deep, and lead to caries, gangrene, periostitis, and tensive contractions and ankyloses. Ulceration, according to *icholls*, occurs in about 8 per cent. of cases.

The condition may become chronic. If, however, the more vourable termination ensues, and absorption commences, the ocess may be completed in about six weeks, if the patient be rong and healthy. In debilitated subjects, the attack may be olonged for months, with successive crops of papules.

The various types and situations of the papules have given rise to ecial nomenclature.

Crab Yaws.—Occurring under the horny tissues of the plantar and lmar surfaces. Much pain and local inflammation is set up in nsequence, and gives rise to ulcerative fissures.

Ringworm Yaws.—A confluent circular ring of papules is so esignated.

Cacca Yaws (or *Pian dartre*).—Applied to a papulo-squamous,

precursive eruption, which may persist throughout the attack even after the disappearance of the general eruption.

The **Diagnosis** should not be difficult. Syphilis should be excluded, as per the differential table given in the foregoing section on *Etiology*.

The **Prognosis** is favourable, except in young infants or in cases of concurrent grave disorders.

The case mortality is only 2·5 per cent.

Pathological Anatomy.—The histology has been worked out by *Unna*, *Charlouis*, and, more recently, in a very able manner, by *M'Leod.*

It is a chronic dermatitis, having a focus in the papillæ, which are enormously elongated, and their blood-vessels dilated.

There is a cellular infiltration of plasma cells, less dense, however, than in the case of syphilis.

There is a large extravasation of polynuclear leucocytes, and marked hyper-keratosis.

The hair and hair follicles are unaffected, but the sweat glands are dilated.

Treatment.—Cleanliness should be secured by daily warm antiseptic baths. Chills should be avoided, and the food should be nourishing and digestible.

Locally the papules should be treated with carbolic acid medications, or tincture of iodine.

Ulcers should be treated on the usual antiseptic lines.

Of internal remedies **potassium iodide** is the most frequently employed, and, to a less extent, mercury.

Both these drugs appear to have a beneficial effect, and this has often been brought forward as an argument for the syphilitic origin of the disease; but, as *Manson* has pointed out, it would be just as logical to conclude a common origin for scabies and pityriasis versicolor, because sulphur is of marked use in the treatment of both!

Prophylaxis should be secured by isolation of the sick and suitable disinfection of infected quarters.

CHAPTER XXXVIII.

YELLOW FEVER.

Definition.—An acute specific febrile infectious disease, limited to certain geographical regions; and characterised by fever, prostration, icterus, and albuminuria.

Synonyms.—*Black vomit*, *Typhus icteroides*, *Fièvre jaune*, *Gelbes Fieber*, *Febbre gialla*.

History and Geographical Distribution.—The earlier history is not known, but the first account of the disease is from the West Indies. An epidemic, reported by *Du Tertre*, in Guadeloupe, in 1635, may possibly have been yellow fever; but certainly this was the nature of the Barbadoes epidemic in 1647. It appeared in Cuba in 1648 (140 years after the first Spanish occupation). It was first reported in Jamaica in 1655; in San Domingo in 1655; in Martinique in 1688; at Vera Cruz, 1690; in St. Thomas in 1793.

Gaps of thirty to fifty years during the seventeenth and eighteenth centuries occurred in all these places, during which the disease was practically absent.

Other *endemic centres* besides the West Indies are—

Mexican Coast.
Senegambia and Guinea Coast of W. Africa.
Brazil.

On many occasions infected ships have visited European ports, but, except in S.-W. Europe, the disease has never spread.

In the eighteenth century Spanish and Portuguese ports were the headquarters of the W. Indian trade. Cases occurred in Cadiz in 1700, 1730, 1741, 1764, and 1780; at Lisbon in 1723, and at Malaga in 1741; but none of these showed any tendency to spread inland or along the coast. There were three great Spanish outbreaks, however, in 1800, 1810, and 1819, which caused a great mortality, and spread not only along the coast, but also inland. There have been four later Spanish outbreaks of a milder type, the last being not on the coast, but at Madrid, in 1878, on the return of troops from Cuba.

The disease has never been observed in Asia or Australia.

Etiology.—Yellow fever is caused by a specific germ, the nature of which is still unknown.

From time to time many microbes have been stated to be the cause of yellow fever. These, which have now only historical interest, are:—

Freire's *Cryptococcus xanthogenicus.*
G. Valle's *Peronospora luteæ.*
Da Lacerda's *Cogumello fungus.*
Sanarelli's *Bacillus icteroides.*

The latter is a short rod, 2 to 4 μ long, with rounded angles and four to eight flagellæ. It is polymorphous, easily stained, and decolourised by Gram. It will grow on the usual media; does not liquefy gelatine; is a facultative anaërobe. It is frequently found in the blood of yellow-fever patients, and the serum of these patients agglutinates the bacillus. It is resistant to desiccation. Inoculation in guinea-pigs, mice, &c., gives a fatal septicæmia.

It is nearly allied to the *Coli* group, but is now definitely known not to be the specific organism of yellow fever.

But whatever the germ may be, the disease is one to which *every race of mankind is susceptible.*

Nor are there limitations in age incidence, except that in *young children the type of disease is extremely mild.*

One attack generally confers immunity which is usually complete.

For many years *Dr. Carlos T. Finlay* of Havana held that the disease could be conveyed by mosquito bite.

Experiments carried out in Cuba, in 1900, by *Reed*, *Carroll*, *Agramonte*, and *Lazear* of the U.S. army, supported this theory of Finlay. Non-immune persons bitten by mosquitoes which had fed on cases two to eight days previously did not develop yellow fever, but, if bitten by mosquitoes which had fed on patients ten to thirteen days before, yellow fever occurred. *Lazear* himself was bitten, developed the disease five days later, and died after six days' illness.

Nature of Mosquito.—It is now definitely settled that the disease is conveyed by the bite of the Stegomyia fasciata.

Other mosquitoes from infected areas—*Culex fatigans*, *Culex confirmatus*, and *Culex tæniorhyncus*—have been investigated, and invariably with negative results.

Our chief knowledge of the etiology of yellow fever is based on the excellent work of the French Mission to Brazil in 1903, consisting of Drs. *Marchoux*, *Salimbini*, and *Simond*, the report of which was published in the *Annales de l'Institut Pasteur* for November, 1903.

Their conclusions, together with remarks by other observers, may well be classified here, and will give a concise and excellent résumé of our knowledge.

1. The incubation period in the human body is from two to thirteen days—probably most often three to five.

2. The disease is carried from sick to healthy by the intermediate agency of the *Stegomyia fasciata.*

3. The distribution of the *Stegomyia* is between the parallels 43° North and South.

4. No other mosquitoes have, so far, been proved to carry the germ.

5. The microbe of yellow fever must be extremely minute; since, in the serum of a patient, the virus will pass (without dilution) through the Chamberland F bougie, but not through the B bougie.

6. It seems not unlikely that the germ may belong to the Genus Spirillum (*vide* p. 60).

7. The serum of a patient is not infective during the incubation period.

8. The serum of a patient is virulent from the first to the third day of the disease; and the *Stegomyia* must bite the patient during that period, or else will not be capable of transmitting the disease.

9. On the fourth day of the disease the blood no longer contains the virus, even if the fever is high.

10. When a mosquito has bitten a patient during the first three days of his illness, it is not until a further period of at least twelve days has elapsed that this mosquito is capable of infecting a healthy person; and the more this period is exceeded the more dangerous does the mosquito's bite become.

11. The bite of an infected mosquito does not necessarily produce yellow fever, and if yellow fever is not produced, no immunity will be obtained against an injection of virus.

12. The bite of two infected mosquitoes may give a grave form of the disease.

13. Most mosquitoes lay their eggs on the eight consecutive days after they have obtained their first bite. The female *Stegomyia* differs in that it does not die after its first batch of eggs has been laid, but lives until it has deposited seven successive batches; the average period of its survival in the mature state being from twenty to thirty days. It is, therefore, capable of transmitting the infection to a large number of individuals after the twelve days' incubation period of the virus within the insect's body is over.

14. Ova laid more than twelve days after the ingestion of infective blood by the parent mosquito would appear to be capable of infecting the imago which springs from such ovum; but such mosquitoes hatched out from infected ova do not acquire the power of conveying infection until after the fourteenth day of their existence in the perfect state.

15. It is found that 0·1 c.c. of serum taken from a patient during the first three days of his illness, will produce yellow fever in a healthy person if injected under the skin.

16. The virus, deposited on a superficial skin abrasion, does not produce the disease.

17. Virulent serum, if exposed to the air at a temperature of 24° to 30° C., is inert at the end of 48 hours.

18. In defibrinated blood kept under vaseline oil at 24° to 30° C., the microbe is still living at the end of five days.

19. At the end of eight days, defibrinated blood kept under the same conditions no longer contains active virus.

20. Virulent serum becomes harmless after an exposure of five minutes to a temperature of 55° C.

21. Such serum which has been rendered harmless by an exposure of five minutes to a temperature of 55° C., if injected into a healthy person, will produce a relative immunity; and, if followed by the inoculation of a very small quantity of virus, this immunity may become complete.

22. The serum of a convalescent possesses definite prophylactic properties, which commence on the eighth day, and are still appreciable at the end of twenty-six days.

23. The serum of a convalescent appears to have therapeutic properties.

24. Contact with a patient, his clothes or excreta cannot produce yellow fever.

Symptoms.—Occasionally prodromal symptoms, such as malaise, headaches, anorexia, giddiness, &c., may be felt for a day or two before the attack.

As a rule, however, the *onset of the disease is sudden.*

A slight rigor, often beginning at night, ushers in an attack of *fever.*

Severe frontal headache and racking pains in the limbs and loins are the usual symptoms of the invasion stage.

The temperature continues to rise, and as the chill passes off the face becomes swollen and red, the conjunctivæ injected, and the eyes have a staring and shining appearance.

The breathing is hurried, and the pulse full and strong.

The tongue is moist and furred, with red edges. The gums are spongy, and bleed readily.

There is intense thirst, and a feeling of oppression and pain at the epigastrium. The stomach becomes irritable, and *vomiting* of a clear acid fluid is usual.

The *bowels* are generally constipated. The *urine* is scanty, of acid reaction, high specific gravity, and has diminished urea. From the second day it is found to contain albumen.

The temperature reaches its maximum in twenty-four to thirty-six hours, and may continue for two or three days (Fig. 77).

At about the third day there is an abatement of the symptoms.

The temperature and the pulse fall; the headache gets better. The injected conjunctivæ give place to a jaundice tint, which may extend to the rest of the body. There is an abatement of the gastric irritability.

This is a crucial point in the disease. If a favourable turn is taken the temperature and pulse fall gradually to normal; the urine increases, and the albumen diminishes until at last convalescence is established.

If, however, the case is a bad one, the gastric symptoms will reappear in an aggravated form, and vomiting of a chocolate-coloured or uniformly black fluid sets in. This is accompanied by a recrudescence of the fever, or, less often, by a sudden subnormal drop. The black vomit seldom appears before the third, and more often on the fifth or sixth, day. It is a dangerous, though not absolutely fatal, symptom.

The sudden drop in the temperature is, however, an ominous sign, especially if the pulse curve mounts at the same time.

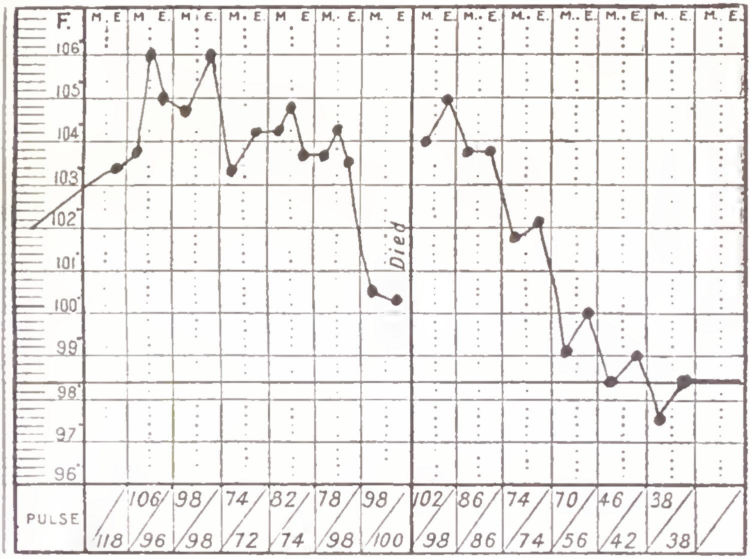
Consciousness is generally maintained, but much apathy and prostration are present.

More rarely, there is much delirium with intense jaundice.

Hæmorrhages in various parts of the body may occur.

The *urine* will be found to be abnormal. Albumen, globulin, and casein are usually present. Creatin is copious; and there is a deficiency of urea and chlorides.

During the first six months pregnant women almost always miscarry, and rarely recover.



Case 1.—Severe case. Death. Case 2.—Mild case. Recovery.

Fig. 77.—Yellow fever. Temperature charts.

Death most frequently ensues between the fourth and tenth day of the disease.

If recovery takes place, convalescence is always protracted.

Complications occasionally prolong the convalescence, such as parotitis, abscesses, boils, gangrene.

Relapses are rare.

As in most diseases, besides the typical cases, other forms appear.

There is an ambulatory type, such as chiefly occurs in children.

There is a fulminating type, with death in the first thirty-six hours.

Mortality.—According to the severity of the disease, the mortality may vary from ½ per cent. in a mild epidemic to 85 in a severe one.

It is much less amongst natives than amongst non-immune whites.

Death is most frequently caused by toxic exhaustion or by uræmia; more rarely by hyperpyrexia or hæmorrhages.

Diagnosis.—Malaria, blackwater fever, and relapsing fever may all have to be excluded.

Although at present the geographical distribution is different, yet septicæmic and early pneumonic plague have many features in common. The author has seen several cases in their early stages with an extraordinary resemblance to plague. The abrupt onset, the high temperature, the frontal headache, the white tongue with red edges, the injected conjunctivæ, the anxious expression are all common to both diseases. In plague, however, there will either be a bubo, or pneumonia or bacilli will be found in the blood. Moreover, the plague pulse, though just as fast and full, is more compressible; the gait is more drunken; and there is not the same gastric irritability nor subsequent jaundice as are found in yellow fever.

Morbid Anatomy and Pathology.—*Rigor mortis* sets in early, and is pronounced. The blood is fluid, and the serum is tinged with yellow by free hæmoglobin.

As a rule, both cutaneous and internal *icterus* will be found, as also cutaneous petechiæ and ecchymoses.

The changes in *the liver* are the most constant and important. It is scarcely enlarged at all, but is soft and of a mottled yellow colour. There is œdema of the intra-lobular connective tissue, and the branches of the portal vein may be hyperæmic; but the whole organ is usually anæmic. Microscopically there is fatty degeneration and cloudy swelling of the hepatic cells.

The *stomach* contains masses of black, thin, or tar-like fluid blood. This black fluid under the microscope will be found to consist of glandular epithelium, mucus cells, deformed red cells, and granular débris, all coloured with hæmatin. The mucous membrane is irregularly congested, and often softened.

The *intestines* may contain the same black fluid with an acid reaction. There is patchy hyperæmia of the small intestine; the solitary glands and Peyer's patches are sometimes enlarged, and Lieberkühn's crypts show fatty degeneration.

The *colon* is generally normal.

The *brain* is frequently hyperæmic, and pronouncedly yellow; surface punctiform hæmorrhages being not infrequent.

The *spleen* is not enlarged or altered.

The *kidneys* show a parenchymatous nephritis. Subcapsular and cortical hæmorrhages are common.

Albuminoid infarcts may be found in the tubules. The capillaries show evidences of fatty degeneration.

Treatment.—This is more a matter of nursing than of drugs.

Either calomel or castor oil should be given at the beginning of the attack; and a hot mustard foot-bath enjoined.

For *hyperpyrexia*, cold pack or sponge should be used, and antipyretics avoided.

For relief of the *vomiting*, a mustard plaster should be applied to the epigastrium, and morsels of ice sucked occasionally; a few drops of spirits of chloroform in a teaspoonful of water are sometimes of use.

In *collapse*, stimulants should be exhibited, subcutaneous injections of ether given, and hot bottles applied generously.

Nourishment should be afforded by iced milk and lime water for the first three or four days; broth and beef tea, &c., or jelly, may then be given, and a slow return to normal diet made.

Routine treatment in mild cases might take the following form:—

℞.—Liq. ammon. acet., ...	ʒij.
Pot. nitratis, ...	gr. v.
Spir. æth. nit., ...	ʒss.
Aq. anisi, ad	℥i.

M. Ft. Mist.

Sig.—℥i. every three hours until the temperature is down to normal.

In more severe cases the following mixture might be tried:—

℞.—Tinct. aconiti, ...	♏ij.
Liq. ammon. acet., ...	ʒij.
Tinct. jaborandi, ...	ʒss.
Aq. cinnam., ad	℥i.

M. Ft. Mist.

Sig.—℥i. every two hours until the urgent symptoms are relieved.

Or, again, to counteract the acid diathesis, the *Sternberg treatment* is of use.

℞.—Sod. bicarb., ...	gr. CL.
Hyd. perchlor., ...	gr. $\frac{1}{3}$
Aq. ad	℥XL.

M. Ft. Mist.

Sig.—℥iss. to be taken every hour, well iced.

Serumtherapy is yet in its infancy. The researches of the French Commission lead us to suppose, however, that an efficient curative or therapeutic serum is within measurable distance of clinical realisation.

Prophylaxis.—Virulent serum which has been killed by heat, or the serum of convalescent cases taken between the eighth and twenty-sixth days, are both found to confer immunity.

It is, therefore, probable that a prophylactic for routine use amongst suspects will be before long available.

In addition to the usual disinfection of soiled articles, &c., all patients should be promptly isolated, and all precautions taken to keep off mosquitoes during the first three days of the illness.

A period of *fifteen days' quarantine* after the last possible chance of infection should be insisted on against all those coming from an endemic or infected area.

General prophylaxis should be secured by enforcing proper municipal cleanliness and sanitation.

An anti-stegomyia crusade should be actively undertaken.

A certain amount may be done, as suggested by *Eyles* in Belize, by isolating each case as it occurs and destroying the mosquitoes in the immediate neighbourhood; but, as *Ross* points out, mild cases may not be recognised, infected eggs are not dealt with, and infected cases introduced by land or water will always be a grave source of danger to a community as long as *Stegomyia* are, like the poor, always with us. A proper campaign is the most rational safeguard, and its feasibility has been demonstrated at Havana, Ismailia, and other places.

Such schemes for the public weal should not be carried out in any half-hearted way.

The accelerated conditions of ocean travel which will be afforded in a few years' time by the opening of the Panama Canal and the consequent linking up of the W. Indian endemic yellow-fever area with the rich virgin material of the further East, may very likely introduce the yellow-fever germ into the crowded and insanitary oriental cities, where *Stegomyia* is perhaps the most common of all mosquitoes.

Such a fatality will need prompt recognition of cases, and swift and thorough action, if we wish to avoid adding another fatal burden to the awful trio of plague, cholera, and smallpox, under which the hapless oriental groans and dies.

CHAPTER XXXIX.

MICROSCOPY.

PART I.—THE INSTRUMENT.

Since 1824, when *Tully* constructed his first Achromatic Microscope, the progress towards perfection has steadily advanced.

Within quite recent years the optics of microscopy have been entirely changed by the introduction of apochromatic objectives (by Zeiss) giving a wide range of magnifying power and a great working distance, which previously had been thought impossible.

The evolution of the microscope has been due in no small degree to the admirable labours of the Royal Microscopical Society and the Quekett Microscopical Club, both of which meet at 20 Hanover Square, and to one of which it is highly desirable that every microscopist should belong.

Not many years ago a hospital rarely possessed more than one microscope, and that was carefully preserved under a glass case as a kind of *objet d'art.* Now, the microscope is part of the *impedimenta* of every serious student.

The essentials for sound work are easily summed up:—

1. A good stand.
2. Good objectives and eyepieces.
3. A practical working knowledge of the instrument.
4. A sound acquaintance with the methods of mounting, staining, and general technique.

The number of excellent instruments on the market is now so great that it would seem almost invidious to make comparisons; but to the student of tropical medicine the subject is of such importance that enumeration of one or two good instruments may not be out of place, and may save both money and anxiety.

To those to whom cost is no consideration, the author would strongly recommend an instrument made by Messrs. C. Baker, and called the "Nelson Model Microscope No. 1, A" (Fig. 78).

It is an almost perfect instrument, costing, without lenses, £35.

The range of movements and mechanical contrivances are without equal in any Continental microscope.

The mechanical stage has not only the usual lateral and forward movements, but also diagonal and rotational. All these movements are secured by heads working on a single axle, thus obviating the movement of the hand from place to place. The fine adjustment is

so placed as to be protected from accidental injury, and is in such a position that the hand can rest on the stand while manipulating it.

There is a fine adjustment also to the substage condenser.

Grooved slots are provided to the feet for firm fixation during

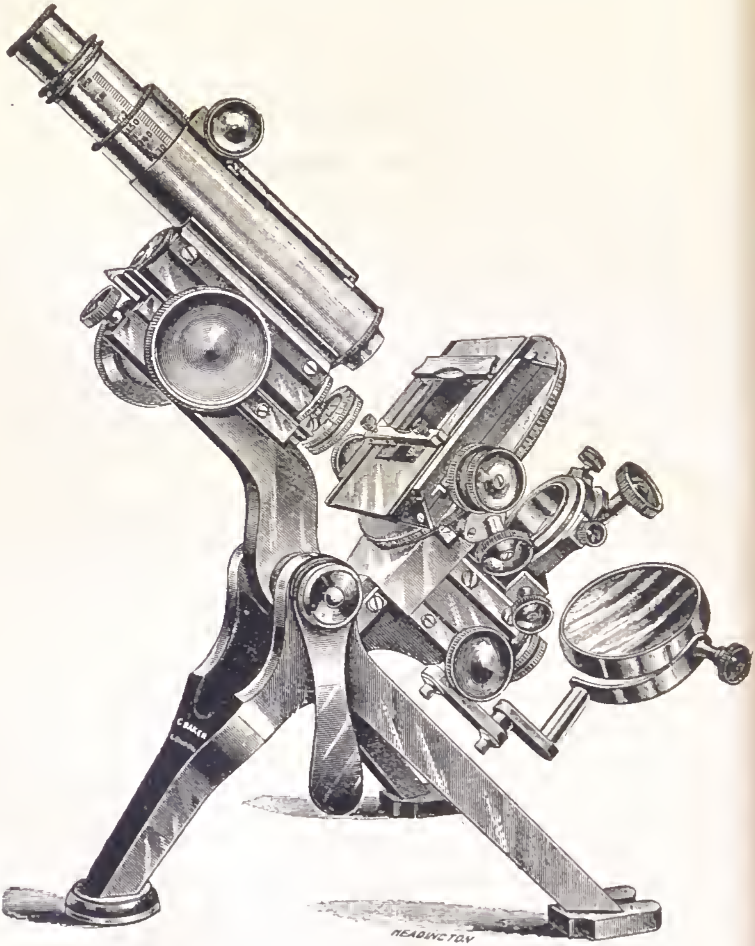


Fig. 78.—Nelson microscope.

micro-photographic work. These and a hundred other points make the instrument of the highest scientific use, and permanent value.

Zeiss' lenses should be used with it.

For those who require a sound instrument for laboratory use, but at less expense than the former, would do well to procure a Leitz's

stand No. B. (Fig. 79) at a cost of (without objectives and eyepieces) £14; and for this again Zeiss' lenses should be used.

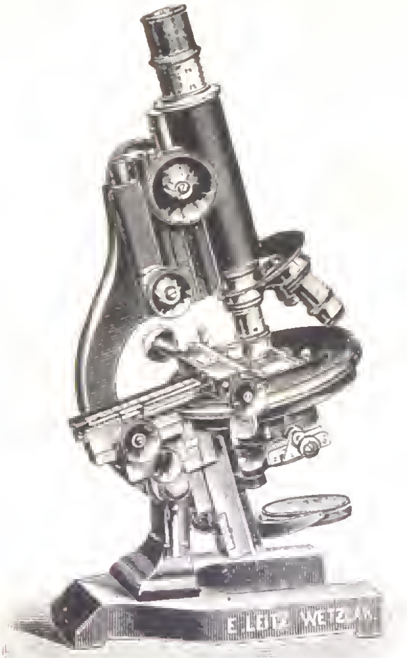


Fig. 79.—Leitz microscope.

The cost of Zeiss' lenses is as follows:—

ACHROMATIC.

Dry Series.

$1\frac{9}{16}$ inch,			£0	12	0
$1\frac{3}{5}$,,			0	12	0
$1\frac{1}{4}$,,			0	12	0
$1\frac{1}{2}$,,	—1 inch,		2	0	0
1 ,,	N.A.,	·17,	1	7	0
$\frac{3}{4}$,,	,,	·20,	1	4	0
$\frac{3}{4}$,,	,,	·30,	1	10	0
$\frac{1}{2}$,,	,,	·35,	1	10	0
$\frac{9}{32}$,,	,,	·40,	1	16	0
$\frac{1}{6}$,,	,,	·65,	2	2	0
$\frac{1}{6}$,,	,,	·85,	2	14	0
‡E $\frac{1}{9}$,,	,,	·85,	3	6	0
‡F $\frac{1}{14}$,,	,,	·85,	4	4	0

WATER IMMERSION.

Plankton Searcher.

	$1\frac{3}{8}$ inch.	N.A.	·11,	. . . .	£1 0 0
D*	$\frac{1}{6}$,,	,,	·75,	. . . .	3 15 0
‡H	$\frac{1}{10}$,,	,,	1·15 to 1·20,	. . . .	5 10 0
‡J	$\frac{1}{14}$,,	,,	1·15 to 1·20,	. . . .	7 4 0

OIL IMMERSION.

$\frac{1}{12}$ inch.	N.A.	1·25,	. . . .	£8 0 0

APOCHROMATIC.

DRY SERIES.

Initial Power.

*24·0 mm.	1 inch.	10·5	N.A.	0·30,	. .	£6 0 0
16·0 ,,	$\frac{2}{3}$,,	15·5	,,	0·30,	. .	4 0 0
*12·0 ,,	$\frac{1}{2}$,,	20	,,	0·65,	. .	7 0 0
8·0 ,,	$\frac{1}{3}$,,	31	,,	0·65,	. .	5 0 0
†*6·0 ,,	$\frac{1}{4}$,,	41·5	,,	0·95,	. .	9 0 0
†4·0 ,,	$\frac{1}{6}$,,	62	,,	0·95,	. .	7 0 0
†3·0 ,,	$\frac{1}{8}$,,	83	,,	0·95,	. .	8 0 0

WATER IMMERSION.

†2·50 mm.	$\frac{1}{10}$ inch.	100	N.A.	1·28,	. .	£12 10 0

OIL IMMERSION.

3·0 mm.	$\frac{1}{8}$ inch.	83	N.A.	1·30,	. .	£15 0 0
3·0 ,,	$\frac{1}{8}$,,	83	,,	1·40,	. .	20 0 0
2·0 ,,	$\frac{1}{12}$,,	125	,,	1·30,	. .	15 0 0
2·0 ,,	$\frac{1}{12}$,,	125	,,	1·40,	. .	20 0 0
1·50 ,,	$\frac{1}{18}$,,	167	,,	1·30,	. .	17 10 0

Compensating Eyepieces.

	No. 2.	4.	4*.	6.	8.	12.	18.	27.
160 mm. tube,	20/-	20/.	40/-	20/-	30/-	30/-	25/-	—
250 ,, ,,	25/-	25/-	—	—	35/-	30/-	30/-	25/-

But there is a class of instrument which may prove even more serviceable still.

Not only in the matter of books has the tropical practitioner need for a *multum in parvo*, but the exigencies of travel also necessitate economy of space, whenever possible, in other directions.

* These three lenses are made for the 10-inch tube only, all the others are supplied corrected for either the long tube or the short tube, as ordered.

† Provided with correction collar.

‡ With correction collar, extra, 20/-

For a compact and admirable instrument the author can recommend *Beck's "London" microscope (portable model)*, Fig. 80, one of which has accompanied him in various parts of the world for the last ten years, and is still as good as ever.

The whole microscope, *with mechanical stage*, triple nosepiece, two eyepieces, and four objectives, fits into a brass-screwed mahogany case, measuring only $2\frac{1}{2}$ inches by $4\frac{3}{4}$ inches by $9\frac{3}{4}$ inches. The advantages of such compactness need no comment. The total cost, complete with objectives, &c., is only £19.

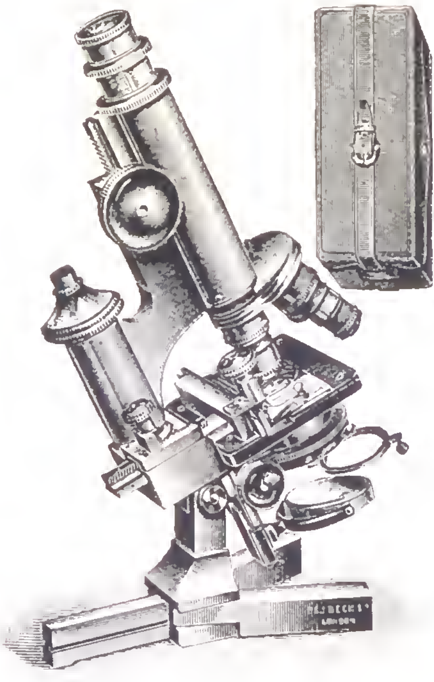


Fig. 80.—"London" microscope.

Zeiss' lenses can be supplied at a small extra cost.

Before leaving the subject of instruments, three pieces of auxiliary apparatus should be mentioned.

A *drawing eyepiece* (Fig. 81) will be found invaluable for parasitic and other work; while, for insect dissections, which form such a large part of modern medical research, *a dissecting microscope*, such as that shown in Fig. 82, will be a matter of necessity. The third piece of auxiliary apparatus, which has been recently introduced (Messrs. R. & S. Beck, Ltd.), is *Gordon's apparatus for photomicrography*, which brings the photographic reproduction

of microscopic results within reach of the busiest practitioner (Fig. 81*a*).

It is a new application of photography to the microscope, which

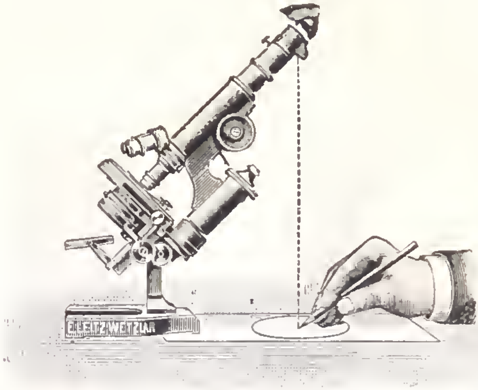


Fig. 81.—Drawing eyepiece.

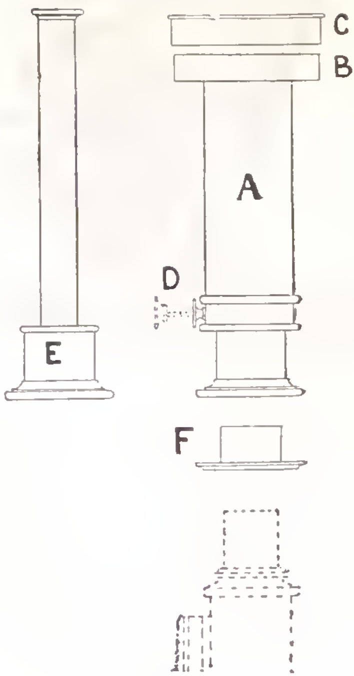


Fig. 81*a*.

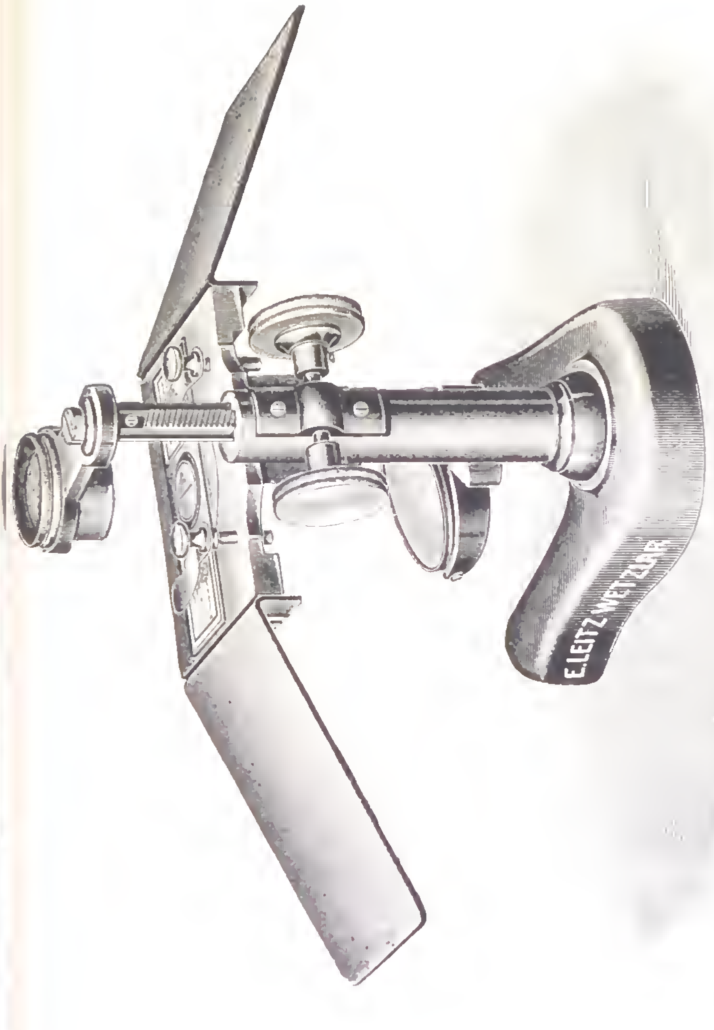


Fig. 82.—Dissecting lens.

renders the process certain and entirely simple. The microscope is used in a vertical position, exactly as usual, and the photographic apparatus may be likened to a photographic eye. It consists of a tube, A, about 6 inches long, which is placed over the microscope eyepiece when a photograph is desired. At the upper end of this tube, B, a small photographic plate $1\frac{5}{8}$-inch square, is held by means of a cap, C, in a light-tight chamber; between this and the eyepiece is a projection lens focussed upon the plate, and a small exposing shutter, D, is placed in the tube for making the exposure.

This small camera can be kept ready with a plate in position, on the observer's table, and all that has to be done if a photograph is desired is to place it on the instrument and make the exposure. If the observer's eyesight is normal the photograph will be sharp when the microscope is in its ordinary focus, but as almost everyone has slight errors of vision of some kind, it has been found desirable to supply a duplicate tube, E, with a focussing eyepiece of high power, which is first placed on the instrument in order to focus, and is then replaced by the camera.

A small flange, F, fitted over the eye end of the microscope, is required to form a table upon which to rest the camera; it may be kept continuously in position on the microscope. With this apparatus photographs can be made $1\frac{3}{8}$-inch in diameter, which are sufficiently large for records and lantern slides, and have such fine detail that they will bear enlargement to any reasonable size.

The most expensive item of a microscopic outfit is the $\frac{1}{12}$-inch oil-immersion lens, usually costing £4, £5, or more. To get a good one is money well spent.

Recently Henry Gowland, optician of Selsey, Sussex, has put on the market a $\frac{1}{12}$-inch lens at a price of only £2 15s. This gives sufficiently good definition to be useful for ordinary work.

The **care of a microscope** is most important.

Never allow dust to accumulate; keep it in its case, or under a glass shade.

The stand should be dusted with a camel's-hair brush, and the lenses wiped periodically with a mixed silk-and-cotton handkerchief softened by many washings.

Unless absolutely necessary, never unscrew the objectives.

If oil has dried on a $\frac{1}{12}$-inch lens, put some fresh oil on it, and allow to stand for an hour, when it should be carefully wiped, and the whole is cleaned off together.

When travelling, prevent undue shaking and jars, by stuffing the case with tissue paper.

Optical Terms used in Microscopy.

Angular Aperture.—This is the angle of the cone which envelopes the pencil of light that is received by the objective from a point on the object.

The efficiency of objectives is expressed by a notation which takes cognisance of the medium surrounding the front of the objective and its influence on the formation of the image. This notation was introduced by Prof. Abbé, and is called the *Numerical Aperture* (N.A.). It stands for the efficiency of an objective to the passage of light pencils included in the formation of the image.

The formula for the N.A. is $n \sin u$.

n = refractive index of external medium.
u = half the angle of aperture.

With dry lenses it follows that the greatest value of the N.A. is unity, corresponding to an angular aperture of 180°.

Aplanatism.—Freedom from spherical aberration (*q.v.*).

Chromatic Aberration.—White light, in its passage through a lens, is decomposed into its component colours in the same manner as with a prism. These colours are refracted from their original course, but the colours at the red end of the spectrum are refracted the least. The result is that, on emergence from the lens, the rays have different foci for the different spectrum colours.

The non-union of these rays in one focal point is termed chromatic aberration.

Negative eyepiece is one for examining an image formed at the diaphragm between the two components of a plano-convex lens. The Huyghenian is the best known.

Refractive Index.—When a ray of light passes obliquely from one medium to one of a different density the path of the ray is refracted.

The ray entering the medium is called the *Incident Ray* (I.R.).

The ray emerging from the medium is called the *Refracted Ray* (R.R.).

The *angle of incidence* is the angle between the I.R. and the perpendicular.

The *angle of refraction* is the angle between the R.R. and the perpendicular.

The refractive index is obtained by dividing the sine of the angle of incidence by the sine of the angle of refraction.

Spherical Aberration.—Rays of light passing through the marginal portion of a lens come to a focus nearer to the lens itself than do the central rays.

The interval between these two focal points is termed the spherical aberration.

In compound lenses this spherical aberration can be corrected for one or more special rays, and a lens so corrected is called *Aplanatic*.

The Illumination of Transparent Objects—*Low Powers.*—The light from a white cloud or lamp should be thrown up by means of the concave mirror through the object, and the Iris diaphragm closed to exclude extraneous light. The mirror must be so arranged as to direct the light along the optic axis. This can be

ascertained by seeing whether the object appears to move laterally when the microscope body is focussed up and down; if it does so, the position of the mirror should be shifted until the object remains stationary during focussing. The diaphragm may be reduced with advantage to a small aperture, and its distance from the stage varied for the better delineation of difficult structures.

High Powers.—For all powers higher than 1 inch, the use of a substage condenser is recommended. It consists of a series of lenses which condenses upon the object a wide-angle cone of light which can be varied in character to alter the angle of the cone of illuminating light. This method should be used for all powers higher than $\frac{1}{4}$ inch, and for lower powers the same principle adopted, the condenser being moved downwards out of focus to illuminate the whole field.

The Illumination of Opaque Objects—I. **The Bull's-eye Condenser**—*Low Powers.*—The principle of all opaque illumination is to throw upon the object a small and very brilliant image of the source of light. With object glasses ranging from 3 inches to $\frac{1}{2}$ inch, the light can be thrown upon the object by means of the bull's-eye or side condenser.

Manipulation.—1. The light should be as near as convenient, about 10 inches.

2. The light, bull's-eye, and object glass should be in line.

3. The light should be above the level of the stage, as high as the object glass will admit, so that the shadows shall not be exaggerated.

4. The bull's-eye should be so placed that a small image of light is focussed on the object.

Remarks.—A large bull's-eye is better than a small one, but it does not make as much difference as is generally supposed, because, although it collects more light, it is generally a less powerful lens and produces a larger image, thus spreading out the light over a larger area.

Character of Illumination, one-sided.—The chief point to be observed is that the light coming from one direction causes heavy shadows and great light and shade contrasts, and only one side of a solid object is illuminated.

High Powers.—A second method of using the bull's-eye enables opaque objects to be examined with powers such as $\frac{1}{6}$ or $\frac{1}{8}$.

Manipulation.—1. The light should be placed slightly below the level of the stage.

2. The bull's-eye should be placed flat surface upwards, slightly above and almost touching the stage.

3. The edge of the lamp flame should face the stage.

Remarks.—A beam of light is reflected from the flat surface and condensed by the curved surface of the bull's-eye into a flat feather in an almost horizontal direction.

Character of Illumination, one-sided.—The shadows will be very marked owing to the great obliquity of the light, and for this reason surfaces to be thus examined must be almost plane.

II. The Side Silver Reflector—*Low Powers.*—By this means a more brilliant illumination is obtained.

Manipulation.—A bull's-eye should be used to parallelise the light.

1. The light should be level with the side silver reflector.

2. The bull's-eye should be centred on the same line.

3. To parallelise the light, the bull's-eye should be so placed that an image of the light is formed on the other side of the microscope on the wall of the room (roughly, about 10 feet away). This is the simplest way of obtaining approximately parallel light.

4. The bull's-eye must be moved, with the light as centre, till the parallel beam of light illuminates the whole surface of the reflector. A white card held in front of the reflector will show the area of its illumination.

5. The reflector is now turned facing the object, and in such a position that the spot of illumination is as small as possible.

The final adjustment is made whilst looking through the microscope.

Character of Illumination.—Brilliant, but one-sided, otherwise like bull's-eye.

III.—The Parabolic Side Silver Reflector—*Low Powers.*—This reflector is best when fitted to the object glass itself.

The light should be taken from the bull's-eye in exactly the same manner as with the side silver reflector. The only adjustment then required is to focus the light on the object by sliding the reflector up or down the object glass mount, and turning its face towards the light.

Character of Illumination.—Maximum brilliancy and many sided. This is the most powerful and best opaque illuminator. It focusses the light more accurately to a point by means of its parabolic shape. It throws light upon the object from a solid 180° of arc out of a possible 360°, and thus illuminates a larger area of a solid object at once. It is very simple to use, as, being mounted correctly on the object glass, it can never be far out of adjustment.

Methods of ascertaining Magnifying Power—*The Magnifying Power.*—To explain magnifying power completely involves the explanation of the elementary theory of the microscope, but the methods of measuring it can be simply described. Once this power of measurement has been obtained, the microscopist can by a few experiments find out that pulling out the draw-tube increases magnifying power, that changing the eyepieces alters it, that the figures given in instrument makers' catalogues are sometimes more and sometimes less accurate, and many other interesting details of like nature.

There are three preliminary points of the utmost importance. The image seen through the microscope is supposed to exist at a distance of 10 inches in front of the eye, no matter where it really does exist. For this purpose one is supposed to see it as if it were a real object placed 10 inches from the eye.

If a microscope magnifies 10 diameters, that image is ten times the size that the object would appear if the object were held 10 inches from the naked eye—not ten times the size that the object would appear if held 5 inches from the eye. A magnifying power of ten means 10 diameters, that is ten times the length of the original and not ten times the area. A magnifying power of × 10 gives an area 100 times that of the object. A floor 10 feet square would contain 100 tiles 1 foot square.

Methods of taking Magnifying Power—*Method A.*—The simplest method. Place a scale or finely-divided rule on the stage of the microscope in the focus of the object glass.

Place one eye to the microscope and place a foot rule upon a book at the side of the microscope opposite the other eye (the unemployed eye), mount the rule up till it is exactly 10 inches from the unemployed eye.

Open both eyes and the scale seen under the microscope, if pushed to foot rule side of the field of view, will appear superimposed over the foot rule, and the size of the two images may be compared.

If $\frac{1}{10}$ of an inch in the microscopic image corresponds with 1 inch on the foot rule the magnifying power is 10, if $\frac{3}{10}$ of an inch corresponds with 2 inches on the rule, the magnifying power will be $2 \div \frac{3}{10} = 6 \cdot 66$, and so on—that is, divide the amount on the foot rule by the amount in the microscope image that corresponds to it.

For anything except low powers a stage micrometer ruled in either $\frac{1}{100}$ or $\frac{1}{1000}$ of an inch, or $\frac{1}{10}$ and $\frac{1}{100}$ of a millimetre should be used, as a scale is not sufficiently finely-divided.

This method is not suited for accurate work because the head must be kept absolutely stationary or one image will move with reference to another. It is, however, convenient for rough purposes.

Having found the magnifying power of an object glass with a particular eyepiece and tube length, the size of an object can always be measured.

Mount a piece of paper exactly 10 inches from the eye, put the object under the microscope towards the paper side of the field of view. Open both eyes and rapidly dot the paper where the two ends of the object appear. This distance divided by the magnifying power gives the actual size of the object.

Method B.—This method is exactly the same as Method A, except that the system is rendered accurate by the use of a Camera Lucida.

By this means, the foot rule or paper, as well as the image in the microscope, are seen at one time by one eye, and therefore they do not move with relation to one another.

The paper or foot rule must be 10 inches from the upper surface of the Camera Lucida Prism, and the eye must be placed as nearly as possible in contact with the Camera Lucida Prism.

If the Abbé Camera Lucida be used, the distance 10 inches must

include the distance between the mirror and the prism, so that the paper must be 10 inches minus that amount from the mirror.

Method C—Wright's Eikonometer.—This instrument is an appliance which can be held over the top of the eyepiece of a microscope without altering the appearance of the image, but it has a scale of divisions which are seen at the same time. These divisions are of exactly the size that a scale of millimetres would appear if they were 10 inches away.

Thus a scale placed in focus in the stage of the microscope will be seen superimposed by the scale of the Eikonometer.

A metric Stage Micrometer must be used; then

$$\frac{\text{The size in mm. in the Eikonometer Scale}}{\text{The actual size in mm. of Stage Micrometer}} = \text{Magnifying Power.}$$

Measurement of Objects.—This can be obtained in three ways:—

1. Vernier adjustment to mechanical stage.
2. A camera lucida, and a stage micrometer.
3. Eyepiece and stage micrometer.

1. A line in the eyepiece can be adjusted to one side of a visual image. The stage is then moved until the other extremity of the object to be measured meets the line, when the stage movement is read off on the vernier. In the absence of other appliances, this method is fairly accurate, and is much used.

2. *Camera Lucida and Stage Micrometer.*—A stage micrometer is a glass slide on which have been ruled a number of lines at intervals of $\frac{1}{100}$ inch or $\frac{1}{1000}$ inch, or else $\frac{1}{10}$ mm. or $\frac{1}{100}$ mm. This is put on the stage like an ordinary object and focussed.

The camera lucida is then fixed to the eyepiece, and the micrometer lines projected on paper and there drawn. A subsequent drawing of an object can thus be compared with the known scale.

3. *The Eyepiece Micrometer and Stage Micrometer.*—A ruled micrometer eyepiece is inserted. The stage micrometer is then put on the stage and focussed. The draw-tube is then pulled out until a certain number of the eyepiece lines are exactly equal to a division (or divisions) of the stage micrometer.

Say that five eyepiece lines will fill $\frac{1}{100}$ inch of the stage micrometer.

The latter is now removed, and the object to be measured is put in its place. Measurements will then be simple, as five of the eyepiece lines (which are still visible) had previously been found equal to $\frac{1}{100}$ inch.

PART II.—STAINS AND STAINING.

Reagents for Staining of Tissues.

Tissue.	Fixing Reagent.	Staining Reagent.	Mounting Medium.
Algæ, &c.	Copper acetate.	Unstained.	Copper acetate.
Blood.	Alcohol and ether.	Leishman.	Balsam.
Brain.*	2 per cent. ammon. bichrom.	Aniline blue-black.	,,
Epithelium.*	2 per cent. pot. bichrom.	Picrocarmine.	Farrant.
Intestine.*	Chromic acid and spirit.	Hæmatoxylin and eosine.	Balsam.
Kidney.*	2 per cent. pot. bichrom.	,, ,,	,,
Liver.*	,, ,,	,, ,,	,,
Lung.*	Chromic acid and spirit.	,, ,,	,,
Lymphatic gland.	Müller's fluid.	Leishman.	,,
Marrow.	Spirit.	,,	,,
Nerve fibres.	Osmic acid.	Osmic acid.	Farrant.
,, trunk.*	Chromic acid and spirit.	Hæmatoxylin and eosine.	Balsam.
Plant stems, leaves, &c.	Spirit.	Hæmatoxylin	,,
Skin.	,,	Hæmatoxylin and eosine.	,,
Spinal cord.*	2 per cent. ammon. bichrom.	Aniline blue-black.	,,
Spleen.*	2 per cent. pot. bichrom.	Hæmatoxylin and eosine.	,,
Starches.	Spirit.	Unstained.	Glycerine jelly.
Yeast.	Camphor water.	,,	Camphor water.

Formulæ for Hardening and Fixing Reagents.

Ammonium bichromate.—Make a 2 per cent. water solution.

Tissue is hardened in three to four weeks.

Transfer to spirit, and change each day till no further colour comes away.

Suitable for nerve tissues.

* If the tissue has been hardened in chromic acid, the section should be placed, before staining, for five minutes in a 1 per cent. watery solution of sod. bicarb. and then washed in distilled water.

Chromic acid and spirit.—Watery solution chromic acid ($\frac{1}{6}$ per cent.), 2 parts.

Add methylated spirit, 1 part.

Tissue hardens in ten days.

Transfer to spirit, and change each day till no further colour comes away.

Suitable for lungs, intestines, &c.

Müller's fluid.

Bichromate of potash,	30 grains.
Sulphate of soda,	15 ,,
Distilled water,	$3\frac{1}{2}$ ounces.

Tissue hardens in three to five weeks.

Transfer to spirit, and change each day till no more colour comes away.

Suitable for lymphatic glands.

Potassium bichromate.—Make a 2 per cent watery solution.

Tissue hardens in three weeks.

Transfer to spirit, and change each day till no more colour comes away.

Suitable for muscle, spleen, liver, and kidney.

Solution of copper acetate.

Acetate of copper,	15 grains.
Camphor water,	8 ounces.
Glacial acetic acid,	20 drops.
Glycerine,	8 ounces.
Corrosive sublimate,	1 grain.

Mix, filter, and keep in stoppered bottle.

Suitable for green algæ, &c., and keeps the colour of chlorophyll for a long time.

Formulæ and Methods for Staining.

I.—SIMPLE STAINS.

Aniline blue-black.—Dissolve 30 grs. of nigrosine in $3\frac{1}{2}$ ozs. of distilled water, then add 1 oz. of rectified spirit, and filter.

Immerse sections for thirty to sixty minutes, wash, dehydrate in spirit, clear in clove oil, and mount in balsam.

Aniline-gentian violet.—Prepare aniline water by shaking 5 c.c. aniline oil and 100 c.c. distilled water. Filter through wetted paper. To 100 c.c. of this aniline water add 10 c.c. absolute alcohol and 11 c.c. concentrated alcoholic gentian-violet. Keep in a stoppered bottle.

Time required for films = two minutes.

Carbol-fuchsin.—Prepare a concentrated solution, thus:—

Fuchsin, ...	1 gramme.
Abs. alcohol, ...	10 c.c.
5 per cent. Aq. sol. acid carbol., .	100 c.c.

For use mix equal parts of this solution and distilled water. This is a valuable stain for quick diagnosis of smears, &c. Put stain on film after fixing by heat, and wash off *immediately*.

Carbol-methylene blue.

Methylene blue, ...	4 grammes.
Abs. alcohol, ...	20 c.c.
5 per cent. sol. ac. carbol., . .	100 c.c.

Time required—two to three minutes.

Eosine.

Eosine, ...	1 grain.
Meth. spirit, ...	1 ounce.

For smears give ½ min.; for sections 5 mins.
Useful as counter stain after hæmatoxylin.

Hæmatoxylin.

Hæmatoxylin, ...	1 gramme.
Alum, ...	10 grammes.
Distilled water, ...	100 c.c.
Alcohol, ...	5 c.c.
Camphor, ...	small piece.

Fit for use after two months, and improves with keeping.
Can be used for some years.
Mix with equal volume of water, and stain for five minutes.
If overstained, decolourise with distilled water 5 parts, acetic acid 1 part.

Leishman.

Solid Leishman (B. W. & Co.),	0·015 gramme.
Methyl alcohol, ...	10 c.c.

This is an excellent and quick stain for blood, as it has the advantages of the staining properties of Romanowsky, and, moreover, the films require no preliminary fixing.

Method.—Place a few drops of the stain on the film for a half to one minute. Add double the amount of distilled water, move the slide to mix it, and allow to stain for five minutes longer, when it can be washed off with distilled water.

Lœffler's methylene blue.—Add to 100 c.c. of a ·01 per cent. solution of potash, 30 c.c. of saturated alcoholic methylene blue.
Stain for five to ten or fifteen minutes.

Osmic acid.—A 1 per cent. solution should be used. Nerve preparations may remain in this for an hour or two.

Picrocarmine.—Rub up 1 gramme of carmine with 10 c.c. of water and 3 c.c. of liq. ammon. fort.; add this to 200 c.c. of a saturated solution of picric acid in distilled water.

Leave exposed to air till it has evaporated to one-third of its bulk; then filter and keep in a stoppered bottle.

Tissues may be stained for half-an hour, and should *not* be washed afterwards. If desired to mount in balsam, the tissue after staining should be dehydrated in a saturated solution of picric acid in methylated spirit. Clear in clove oil, and mount.

II.—COMPOUND STAINS.

Adamson (for hair and skin fungi).—1. Potash (10 per cent.) twenty minutes.

2. Wash in 15 per cent. alcohol.

3. Stain aniline-gentian violet thirty minutes.

4. Without washing (after pouring off the stain) put on Gram's iodine solution three minutes.

5. Decolourise in aniline oil thirty minutes.

6. Counterstain in eosine one minute.

Gram.—Solutions required:—(*a*) Aniline-gentian violet.
(*b*) Iodine 1 grm.: KI 2 grms.: Aq. 300 c.c.

Method:—1. Stain with (*a*) five minutes.

2. Treat with (*b*) ½ to 2 mins.

3. Decolourise in abs. alcohol till no more colour comes away.

4. Wash in xylol.

5. Dry and mount.

Neisser's (for diphtheria).—Solutions required:—

(*a*)	Methylene blue, . . .	1 grm.
	Abs. alcohol, ...	20 c.c.
	Glac. acetic acid, . . .	50 c.c.
	Distilled water, ...	930 c.c.

Filter.

(*b*)	Bismarck brown, . .	2 grms.
	Distilled water, ...	1000 c.c.

Filter.

Method:—1. Stain in (*a*) for one minute.

2. Wash.

3. Stain in (*b*) for one minute.

4. Wash, dry, and mount.

(*Note.*—The true diphtheria bacillus will be brown with blue ends.)

Pittfield's (for flagella).—Solutions required :—

(*a*)	Satd. alc. gentian violet, . .	1 part.
	10 per cent. sol. potash alum. .	10 parts.
(*b*)	10 per cent. sol. acid. tannic.	

Method :—1. Before use mix equal parts (*a*) and (*b*).
2. Flood coverslip with mixture.
3. Warm till steaming. Leave for five minutes.
4. Wash, dry, and mount.

Romanowsky.—Solutions required :—

(*a*)	Methylene blue, . .	1	part.
	Sodium carbonate. . . .	0·5	,,
	Water, ...	100	parts.

Incubate by fire, or in sun for two or three days, until purple colour is noticed at edge of liquid.

(*b*)	Eosine, ...	1 part.
	Water, ...	1,000 parts.

Method :—1. Dilute (*a*) and (*b*) to 1 in 20.
2. Mix equal parts of dilutions.
3. Apply to slide for ten to thirty minutes.
4. Wash, dry, and mount.

Spore-staining.—Method :—1. Stain with hot dilute carbol fuchsin for five or ten minutes.
2. Give rapid plunge in 5 per cent. H_2SO_4 and wash.
3. Carbol.-methylene blue $\frac{1}{2}$ min.
4. Wash, dry, and mount.
(*Note.*—Spores are red ; bacilli blue.)

Syphilis stains.—1. Fix the smear for a moment with osmic acid vapour.
Stain for ten minutes in a fresh 5 per cent. watery dilution of *Giemsa's stain—i.e.*,

Azur II. eosin.	3·0 grammes.
Azur II., ...	0·8 gramme.
Glycerine, ...	250·0 c.c.
Methyl alcohol, ...	250·0 c.c.

Wash in distilled water and dry.
(The *Spirochæte pallida* is reddish-blue, other spirochœtes and bacteria are blue.) Or,
2. Fix the film in absolute alcohol.
Stain for thirty minutes with the following :—

Giemsa's stain.	10	drops.
Distilled water, ...	50	,,
Pot. carb. (1 in 100), . . .	2	,,

Wash in distilled water and dry. Or,

3. Fix the film in alcohol.

Stain for sixteen to twenty-four hours with a 1 in 1,000 solution of Nile blue B R.

(The *S. pallida* is stained dark blue.)

Note.—The parasite of syphilis (the *Spirochæte pallida*) is described thus by Schaudinn. A spirillum or spironema, 10 μ long; very thin, and has 8 to 26 corkscrew spirals. The ends are sharply pointed, and each has one flagellum. It preserves its spirals both in rest and in motion. There is no undulating membrane.

Ziehl-Neelsen (for tubercle).—Method:—1. Stain in hot dilute carbol fuchsin for five or ten minutes.

2. Decolourise in spirit till no more colour comes away.
3. Plunge into 25 per cent. H_2SO_4.
4. Quickly wash in water.
5. Stain ½ min. in dil. carbol-methylene blue.
6. Wash, dry, and mount.

The Mounting of Insects.—The following method of mounting a whole insect in balsam, with pressure, will be found most admirable (*Cross* and *Cole*).

1. Preserve indefinitely in spirit.
2. Soak in water three or four hours.
3. Place in 10 per cent. caustic potash until soft (either hours, days, or weeks, as required).
4. When soft, remove and wash in water.
5. Soak in concentrated acetic acid for twelve hours.
6. Wash in water.
7. Arrange insect in required position on a slide, and keep in position by a second slide approximated to the first, but kept off slightly by side slips of paper. Tie the two together thus.
8. Keep in methylated spirit twelve hours.
9. Untie the glasses; push specimen into a saucer of spirit.
10. Clear in clove oil.
11. Soak in xylol.
12. Mount in balsam.

For smaller parasitic insects.—The above process may damage their structure. They may, however, be mounted whole by the following method:—

Melt 2 ozs. solid carbolic acid, and add ½ dram glycerine.

Soak the insect in this until transparent, which may take any time from an hour to a week or more.

Wash in spirit: allow to soak in xylol, and mount in balsam.

CHAPTER XL.

PHOTOGRAPHY.

PHOTOGRAPHY is such a valuable handmaiden to medical work that it should be taken up by as many medical men as possible; records of new pathological conditions, micro-photographs, &c., collected in many parts of the world, being frequently of extreme value to the scientific community. The Royal Geographical Society are always thankful for photographs of geographical or ethnological interest from any place.

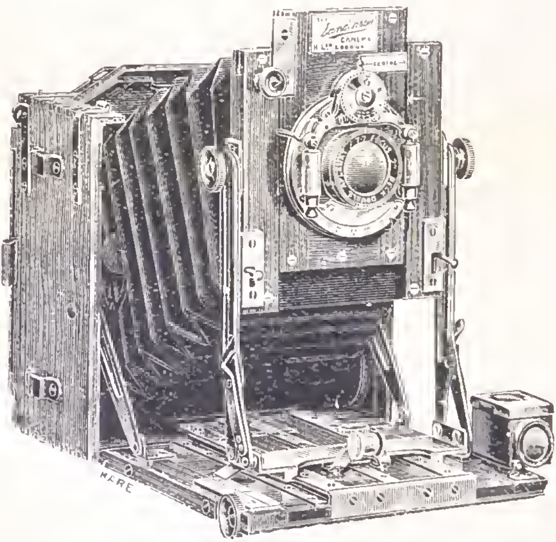


Fig. 83.—Sanderson hand camera.

The following hints are the outcome of the author's own tropical experience in India, the West Indies, and Malaya.

The work does not present nearly all the difficulties that might be expected. The plant need not be cumbersome, nor the manipulations laborious.

Camera.—Nearly all makers now cater for the tropical traveller. The camera should not be too small ($\frac{1}{4}$-plate is rather inferior). Nor should it be too big, or the matter of space and weight will make it a nuisance.

Probably 5 × 4 will be found the best working size for all-round convenience and use. The weight is not excessive; it is large enough to give plenty of detail: it can be conveniently used either as a hand or stand instrument; it is not too large for micro-photography, nor is it too large for direct lantern-slide making.

For the tropics it should be made of teak, brass finished and brass screwed. No glue should be used in its construction, the bellows being screwed to the frame with brass bands.

The lens should be by a good maker, and should give good definition to the edge of the plate with full aperture.

If expense is no object, one of Newman and Guardia's instruments would be found most excellent.

The accompanying cut of the "Sanderson" tropical model, Fig. 83 (made by Messrs. Houghtons, Ltd.), will give some idea of a cheaper but very efficient camera made in strong teak and well suited for rough or tropical usage. The author has had one of these in use for some time, and never found it wanting.

Leather-bound cameras are not suited to the tropics. The glue melts, and insects, &c., eat the leather.

In many tropical places, aluminium is useless; and it will be found better always to have brass fittings in preference. The reason is that the aluminium rapidly oxidises, and the powdered white oxide which results is a poor return for money expended.

Outfit.—The essentials for turning out good practical work are absurdly small:—

1. Case containing camera, lens, slides, &c.
2. Tripod stand (folding).
3. Four vulcanite dishes.
4. Two light metal printing frames.
5. A small red lamp.
6. A bottle of rodinal.
7. 2 lbs. of hypo.
8. Some packets of plates.
9. Some packets of paper (glossy or matt).

Items 3 to 9 will easily go into an ordinary tin deed-box.

Dark-room.—Except when it is required to change plates in the day time (which could always be obviated by having extra slides in the outfit) all the processes can be done without any dark-room. For some ten years the author has practically never used the ordinary hot and ill-ventilated chamber haunted by spiders and cockroaches, and usually designated by that term.

Work can be done at night in most houses. The dining-room table is a useful place, and with a punkah working is always cool.

Except on moonlight nights, or when living in the heart of a town, a verandah will usually answer admirably.

Developing.—Chemicals are apt to deteriorate in hot climates, and weighing out stock solutions is a nuisance.

Burroughs & Wellcome put up convenient photographic tabloids, but a multiplicity of bottles means weight and space, and, moreover, the tabloids have to be crushed and dissolved. The use of a stable one-solution liquid developer is, therefore, highly advisable. Such an one is *rodinal*, which is highly concentrated, and will keep for several years; it is not expensive, and all that is needed for use is to pour out 20 drops (exact amount not highly important) in a wineglassful of water, giving about 2 per cent. solution. This is enough to develop four plates (5 × 4). If under exposed, add a little more water; if over exposed, add a pinch of pot. bromide. Ordinary tap water is quite good enough for use. The following amounts can be used:—

For *Bromide papers*, 1 per cent. solution.

For *Gaslight bromides*:—Rodinal, 1; water, 25; pot. brom., 10 per cent. (three drops to each ounce).

For *Lantern slides*:—Rodinal, 1; water, 30.

The advantages of such simplicity are obvious.

Plates.—Most ordinary makes of plate can be used, and will prove successful if the plates are new. Wratten and Wainwright's are rather more suitably packed for warm climates than are other makes, and keep better in consequence. They should be kept in as cool and dry a place as possible, preferably a drying chamber or bottle, when they will keep good indefinitely.

Paper.—This also should be kept in a drying chamber or bottle, but even then will not keep good for long.

Any gelatino-chloride, P.O.P., or albumen paper may be used, but a collodio-chloride is better for the tropics.

A useful paper which saves much trouble is the "Paget self-toning paper" made by the Paget Prize Plate Co., Ltd., of Watford, England. There is a certain amount of gold in its sensitised surface, thus doing away with the necessity for toning. All that is required is to wash the print in some salt and water, and then fix it as usual in hypo. This gives a good dark sepia tone.

If a blacker tone is required, it can either be toned in the ordinary way like any P.O.P., or in the following platinum bath.

Stock—

Potassium chloro-platinite	gr. 15
Sodium chloride, ...	gr. 150
Citric acid, ...	gr. 150
Water to	7½ ozs.

For use take 1 part of this to 10 of water. The print should be previously washed for ten minutes in a saline bath (salt 1 oz., water 10 ozs.). The paper can be obtained with either smooth or matt surface, the glossy being the better for scientific detail.

For *Bromide Paper* work in warm climates, one of the best is "Kruxo" made by the Hilborn Photo Paper Company, Cedar Rapids, Iowa. It is suitable for either daylight or lamplight exposures.

Formulæ and Tips.

Backing to prevent halation—

Gum arabic solution, ...	1 oz.
Caramel, ...	1 ,,
Burnt sienna (ground in water), . .	2 ozs.
Mix and add alcohol	2 ,,

Blackening for aluminium.—Cleanse the metal with fine emery powder, wash, and immerse in—

Ferrous sulphate, . . .	1 oz.
White arsenic, ...	1 ,,
Hydrochloric acid, . . .	12 ozs.
Dissolve and add water, ...	12 ,,

When colour is deep enough—dry and lacquer.

Blackening for wood—

Borax, ...	30 grs.
Glycerine, ...	30 drops.
Shellac, ...	60 grs.
Water, ...	8 ozs.

Boil till dissolved and add—nigrosine W.S. 60 grs.

Brass, to clean.—To 10 ozs. water add 1 oz. nitric acid. Stir and add slowly by drops 1 oz. sulphuric acid.

Remove grease with dilute ammonia—Immerse in above pickle for a short time. Remove and rub with soft cloth.

Development factors

Quinol, ...	5
Imogen, ...	6
Glycin, ...	7
Eikonogen, ...	9
Ferrous oxalate, ...	10
Ortol, ...	10
Paramidophenol, ...	16
Amidol, ...	18
Metol, ...	30
Rodinal, ...	40
Diamidophenol, ...	60

Faded Prints, to restore.—Unmount the print. Immerse in the toning bath.

Gold chloride, ...	1 gr.
Phosphate of soda, ...	20 grs.
Tepid water, ...	20 ozs.

If this does not restore sufficiently, wash and immerse in saturated solution of mercuric chloride.

Wash again and immerse in ammonia solution (10 drops to the oz.).

Fixing Baths (negatives).

1. *Normal*—

Hypo,	4 ozs.
Water,	20 ,,

2. *Acid*—

Sodium sulphite,	$\frac{3}{4}$ oz.
Sulphuric acid,	35 drops.
Hypo,	4 ozs.
Water,	20 ,,

3. *Alkaline*—

Sodium sulphite,	$\frac{1}{4}$ oz.
Sodium carbonate,	1 dram.
Water,	20 ozs.

Hypo Tests—

Potassium permanganate,	2 grs.
Potassium carbonate,	20 ,,
Distilled water,	40 ozs.

A few drops of this rose-coloured solution will turn to greenish-yellow or brown when added to water containing hypo.

Labelling Bottles.—For acid bottles an ink may be made.

Asphaltum,	1 part.
Turpentine,	5 ,,

Dissolve and add lamp black to suitable consistency.

For other bottles dissolve red or black sealing wax in methylated spirit, and use as ink.

Lacquering Brass.—Clean as previously directed. Heat the brass till too hot to hold. Apply one of the two following lacquers thinly with a flat brush. Dry and apply a second coat.

(*a*)	Turmeric,	3 drams.
	Saffron,	8 grs.
	Hot alcohol,	10 ozs.

Shake, decant, and add—

	Gamboge,	3 drams.
	Gum sandarac,	1 oz.
	Gum elemi,	1 ,,
	Dragon's blood,	$\frac{1}{2}$,,
	Seed lac,	$\frac{1}{2}$,,
Or (*b*)	Gamboge,	$\frac{1}{2}$ dram.
	Shellac,	$\frac{1}{2}$ oz.
	Alcohol,	10 ozs.

Mounting solution—

Nelson's No. 1 gelatine, . .	4 ozs.
Water, ...	16 ,,

Dissolve and add—

Glycerine, . . .	1 oz.
Methylated spirit, . . .	5 ozs.

Stripping Films.—Glass negatives are heavy to carry about. The films may thus be stripped without distortion :—

(*a*) Put negatives for two minutes in—

Glycerine, ...	1 part.
Formalin. . . .	10 parts.
Water, ...	20 ,,

(*b*) Dry, and with a sharp knife cut the film all round the edge, down to the glass.

(*c*) Immerse for five minutes in—

Hydrochloric acid, .	½ part.
Rectified spirit,	6 parts.
Water, ...	4 ,,

(*d*) Transfer to water, and strip carefully.

(*e*) Slip under the film (while floating in the water) a clean glass previously coated with : Collodion q.s., Castor oil 5 drops to the oz.

(*f*) Dry in sunlight or wind.

(*g*) Coat the exposed side of the film with the above collodion varnish, and dry.

(*h*) The film negative can now be stripped from the glass and will be found varnished with collodion on both sides. It can then be trimmed and stored.

Toning Baths—

1.	Water, 20 ozs. ;	Sod. phosph., . .	20 grs. ;	Gold,	1 gr.
2.	,, 20 ,,	Amm. sulphocyan.,	20 ,,	,,	2 grs.
3.	,, 20 ,,	Borax, ...	1 dram ;	,,	2 ,,
4.	,, 20 ,,	Sod. acetate . . .	1 ,,	,,	2 ,,
5.	,, 20 ,,	Sod. carbonate, .	5 grs. ;	,,	2 ,,

Weights, Makeshift—

Penny,	weighs	145	grs.	or	9·5 grammes.
Halfpenny,	,,	88	,,	,,	5 ,,
Threepenny piece,	,,	20	,,	,,	1·2 ,,
Sixpence,	,,	40	,,	,,	2·5 ,,
Shilling,	,,	80	,,	,,	5 ,,
Florin,	,,	160	,,	,,	10·3 ,,
Half-Crown,	,,	200	,,	,,	13 ,,

Three pennies weigh 1 oz. avoird. A halfpenny is just 1 inch in diameter.

Waterproof solution for wood—

Asphalt,	4 ozs.
Rubber,	30 ,,
Naphtha,	10 ,,

Apply with stiff brush and give three successive coats, allowing each to dry. Beware of inflammable vapour.

CHAPTER XLI.

DISINFECTION

This subject is one of the highest importance, as well as of the widest application. Measures against the diffusion of infection should be founded on our knowledge of the natural history of the various diseases. The scheme of procedure will naturally be quite different for each of such different infections as plague, cholera, or smallpox.

Speaking generally, application is usually required in three different types of circumstances:—Shore disinfection of a house, a room, persons, furniture, or effects; ship disinfection of holds, cargo, tanks, store-rooms, passengers, or luggage; or disinfection of large bodies of men, such as troops, emigrants, immigrants, or prisoners.

An admirable example of the latter type has been furnished by the Japanese, who, at the close of the Russo-Japanese War, in an incredibly short time completely disinfected 800,000 returning troops at four different quarantine stations. Their fore-thought and procedure are an object-lesson to occidental nations. The disinfecting staff boarded each transport as it arrived, and every article not carried on the person was disinfected on board before landing. Each man in landing passed through the quarantine station, where he had a hot sea-water bath (50° C.), his clothing, equipment, and arms being meanwhile subjected to steam or formalin disinfection, as the case demanded.

For purposes of this chapter, the subject will be divided into two heads:—

I. Classification of disinfectants.
II. Application of disinfectants.

I. CLASSIFICATION OF DISINFECTANTS.

1. PHYSICAL AGENTS.

(*a*) **Sunlight.**—Sunlight is an active germicide, destroying spores as well as bacteria. The germicidal power, however, is only possessed by the blue, violet, and ultra-violet rays, and is dependent on their intensity.

Röntgen rays have no such effect.

The critical time required varies with the different organisms. The following are some approximate times:—

Bacillus coli communis (in water), .	1 hour.
Anthrax cultures, ...	$2\frac{1}{2}$ hours.
,, dry spores, . . .	8 ,,
Typhoid, ...	6 ,,
* Diphtheria, ...	24 ,,
Tubercle, ...	7 ,,
Plague, ...	$\frac{1}{2}$ hour.
Cholera, ...	1 ,,

(* In diffused light.)

The action of sunlight is only on surfaces directly exposed to the rays, and is probably of a chemical nature.

(*b*) **Electricity.**—The currents themselves seem to have but little bactericidal action; and the effects are probably due either to the heat generated or to electrolytic action.

Hermite has experimented with sewage sterilisation. Sea water was added to the sewage, and the electric current converted much of the chloride into hypochlorite—an active disinfectant.

The results obtained by X rays are not due to direct germicidal properties, but probably to the production of ozone, hypochlorous acid, &c.

(*c*) **Fire.**—This perfect purifier has but limited application in practical disinfection. Articles of small value, however, should always be treated thus; and it is the best means for the disposal of garbage and refuse.

(*d*) **Dry Heat.**—A temperature of 150° C. applied during one hour will destroy all bacteria; even resistant spores.

Non-sporing bacteria, such as plague, cholera, diphtheria, pneumonia, &c., are destroyed by a lower temperature (110° C.); but the dry heat lacks penetration.

Temperatures not exceeding 110° *C.* (230° *F.*) *will not injure most fabrics.*

(*e*) **Boiling.**—Boiling water at 100° C. for half an hour will destroy all known bacteria and spores. It is better to give two hours' exposure to such resistant organisms as anthrax, tetanus, &c.

The addition of borax or of an alkaline soap increases its penetrating power, and prevents rusting or injury to the cutting edge of sharp instruments.

It is applicable to the disinfection of linen, crockery, and cutlery.

(*f*) **Steam.**—This is one of the most valuable disinfecting agents we possess.

It is quick, reliable, and of great penetration. Bacteria are killed instantly, and spores in a few minutes.

It may be either used streaming, or under pressure.

Streaming steam acts as boiling water, and half an hour's exposure will kill resistant spores.

At a pressure of 15 lbs. the temperature is about 120° C., and should sterilise in twenty minutes. At 20 lbs., with a temperature of 125° C., fifteen minutes should be sufficient.

Steam should *not* be used for leather, fur, skins, or rubber.

If salt be added to the water the boiling point is raised, and steam is given off at a higher temperature than 100° *C., which is highly advantageous.*

Various kinds of steam apparatus are in use, the two chief types being—Thresh's, in which the jacket forms the boiler, and the steam is streaming; and the Nottingham and Washington-Lyons types, which first produce a vacuum and exhaust the air from the interstices of material, and then provide steam at a pressure of 20 lbs. In the former no separate boiler is required.

2. GASEOUS AGENTS.

(*a*) **Formaldehyde Gas.**—This gas (HCOH) is one of the most generally useful and best disinfectants we have. It is an admirable germicide: is non-poisonous: is not destructive; and is a true deodorant.

Practically the only agent which can be used to disinfect the richest apartments without injury: to objects of art or value it has in consequence a wide application.

It has no detrimental effect on cotton, linen, silk, or wool. It does not damage colouring matter, except—to a slight extent—the anilin lavenders. *When heated, a watery solution attacks steel and iron*, but the dry gas has no effect on these metals: and other metals are unaffected either by gas or solution.

It unites readily with nitrogenous products of decomposition and fermentation, forming odourless and sterile new compounds.

Corrosive sublimate coagulates albumen, and is, therefore, practically useless as a disinfectant for every-day use; formalin, on the other hand, combines chemically with nitrogenous organic matter, such as albumen, and therefore has an unlimited application as a potent germicide.

It has a further advantage in that it kills dried organisms quite as well as moist ones.

Weak atmospheric dilutions of the gas—sufficient to kill germs—have but slight effect on mammalian animals.

Guinea-pigs, rats, mice, &c., are not killed after half an hour's exposure to the concentrated gas. There is, however, violent respiratory irritation, which may be subsequently fatal to them.

Its disadvantages chiefly lie in the fact that its penetration power is slow and diffusion poor. The meshes of fabrics tend to polymerise the gas and deposit it as paraform. Moreover, it is not an insecticide; cockroaches, bed-bugs, and such vermin are unharmed by it; mosquitoes, however, are killed by exposure to the gas at ordinary strengths.

Formaldehyde exists in three states:—

(1) CHOH *Formaldehyde gas.*

(2) CHOH
  |
CHOH

Paraform.—A white unctuous solid, soluble in water and alcohol.

(3) CHOH
  |
CHOH
  |
CHOH

Trioxymethylene.—A white powder with a strong odour of the gas, and only slightly soluble in water and alcohol.

In commerce, the solution known as *Formalin* is water containing 40 per cent. of formaldehyde gas.

In practice, the solutions are found to be almost all acid—due to

Fig. 84.—Portable disinfector.

formic acid (HCOOH). They also contain usually about 10 per cent. of methyl alcohol, added to increase the solubility and stability.

Methods of Use.—In using formaldehyde, temperature and moisture are both important factors. Owing to possible polymerisation, disinfection should never be carried out if the temperature is under

15° C. All temperatures over 25° C. aid the disinfecting power of the gas.

The full disinfecting power of the gas is only obtained when the air is saturated; over 75 per cent. of moisture, however, is a good working amount.

The evolution of the gas may be brought about in four ways:—

(1) *Boiling a Formalin Solution.*—For this numerous forms of apparatus, such as autoclaves, retorts, &c., have been devised. One of the best applications is "Thresh's Portable Emergency Disinfector" (Fig. 84), which is a movable box-chamber under which a tray of solution is vaporised with a lamp, and the gas and steam enter and disinfect articles inside.

(2) *Heating Paraform.*—Paraform can be procured in tablets. Twenty or thirty of these are placed in some such receptacle as an "Alformant." A spirit flame beneath the vessel causes the paraform to evaporate completely.

(3) *Spraying.*—As the formalin solution evaporates, the liberated gas has an effect on the article sprayed.

(4) *Method of Walker.*—This is admirable for small rooms, cabins, &c. Five pounds of commercial alum are dissolved in 1 gallon of hot water, and this is mixed with 3 gallons of formalin.

When required for use, 8 ozs. of the above mixture are allowed to drip slowly on to 1 lb. of quicklime, this amount being sufficient for 1,000 cubic feet of space.

The quicklime automatically absorbs the water and liberates the formaldehyde. The alum converts the lime into insoluble calcium sulphate, thus preventing secondary reactions between the lime and the formaldehyde.

(*b*) **Sulphur Dioxide** (SO_2).—This gas is heavier than air and diffuses but slowly. Without moisture it has no bactericidal effect. Cold water takes up thirty times its volume of SO_2, forming H_2SO_3, which is the real disinfecting agent.

The *advantages* are that it destroys vermin, such as rats, mice, flies, fleas, mosquitoes, &c., as well as bacteria. It is also cheap and easily procurable.

Its *disadvantages* are that it does not kill spores, and, therefore, is useless for anthrax, tetanus, &c., and also that in the presence of moisture it bleaches vegetable colouring matter, many aniline dyes, attacks most metals, and weakens cotton and linen fabrics.

In Practice.—One pound sulphur gives 1·15 per cent. SO_2 in 1,000 cubic feet of space. At least 5 per cent. of SO_2 should be present to kill non-sporing organisms in twenty-four hours' exposure.

There are three methods:—

(1) *The Pot.*—This is easy and cheap. Not more than 30 lbs. should be put in each iron pot, standing in water. It should not be heaped up in the middle or will be found hard to burn. Make a hollow crater in the centre saturate with alcohol, and light.

(2) *Liquid* SO_2.—This is expensive. It is sold in tins with about 20 ozs. condensed into a liquid by pressure. The leaden nozzles of several tins are cut simultaneously and quickly inverted into an iron pot, and the operator makes a quick exit.

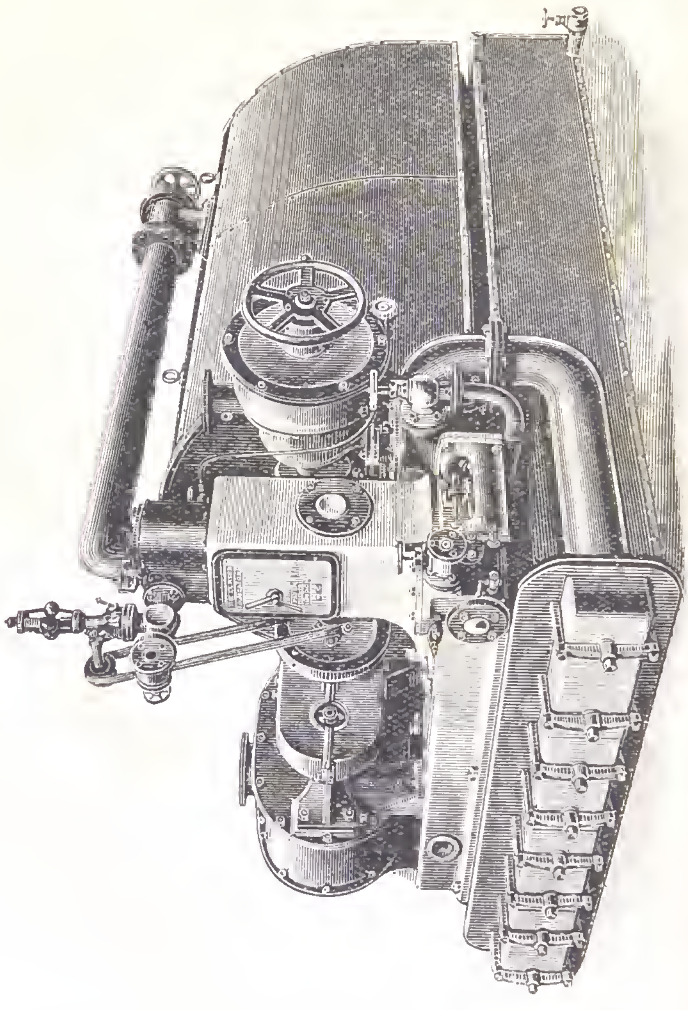


Fig. 85.—Clayton disinfector.

(3) *Sulphur Furnace.*—The best apparatus is the "Clayton" disinfector (Fig. 85).

Sulphur is consumed in a furnace, and the SO_2 removed by a centrifugal fan through hoses. Another hose automatically removes the air

from the infected chamber and passes it back over the burning sulphur. By this means a pressure of gas can be obtained, and the method is admirably adapted to the disinfection of ships' holds and destruction of rats, &c.

Instructions to Officers preparing Vessels for Fumigation by Clayton Process.

For the effectual fumigation of a steamer or vessel, it is necessary to divide her into convenient sections, each of which will be fumigated separately, and such sections should be agreed upon in consultation with the fumigating engineers.

Each hold, between bulkheads, is generally a separate section, and to prepare same watertight doors should be closed, and ventilator cowls covered with canvas. One corner of hatch, or two port-holes, to remain open for the insertion of pipes.

Cabins, saloons, store-rooms, forecastle, &c., also have, as a rule, to be fumigated separately. Lockers, cupboards, &c., should be left open, and all port-holes, skylights, ventilators, &c., closed or covered, to make same as nearly airtight as possible. In the case of alleyways with open ends, and with cabins, galleys, &c., on either side, it is necessary to have the open ends stopped with tarpaulin or canvas, and made as tight as possible.

Any dry stores can be left in store-rooms, &c., while fumigation is going on, but meat, vegetables, soap, and all broken stores required for immediate use, such as tea, sugar, salt, matches, tobacco, &c., should be removed.

It is necessary to remove any carpets, curtains, or coloured cloth stuffs, which may possibly be damp, in which case there is risk of colours being bleached by the action of the gas.

The gas will tarnish bright steel, brass, and other metals, but the same can easily be cleaned and are not damaged thereby. To save trouble in cleaning, electro-plated ware and fittings, and bright metal-work in saloons, state-rooms, &c., should be given a thin coating of common whiting and water, mixed into a paste and allowed to dry on, and removed after fumigation by means of a dry cloth. It is, however, advisable to remove such articles as watches, jewellery, pen-knives, and coins or similar articles from clothing; also keen cutlery, such as razors, surgical instruments, mechanics' tools, &c.

Clothing may be left in lockers or drawers during fumigation, but special care should be taken to see that they are quite dry. The articles should afterwards be brought on deck in order that the air may remove any smell left by the gas. Wet or damp bedding should always be removed if possible.

When the vessel is opened up after fumigation, as much ventilation as possible should be given to all sections. Windsails into holds will greatly accelerate the clearance of the gas. The fumigating machine is provided with a ventilating attachment, which can be used if desired.

The machine is made by the Clayton Co., 22 Craven Street, W.C., and costs about £1,000. £1 worth of sulphur will produce 60,000 cubic feet of SO_2.

The author has had one in use for several years in the large port of Singapore. It is fitted in a steam lighter and can be taken alongside vessels. It answers well, and with care will give 10 per cent. of SO_2.

The annexed is a cut of type "B."

(*c*) **Chlorine** (Cl).—This has considerable germicidal effect, but is uncertain, poisonous, and destructive.

It is heavier than air, and combines with the hydrogen of water in the presence of light, liberating nascent oxygen, to which its disinfecting virtues may be due. It bleaches nearly all organic matter, and has, therefore, a very limited application.

In practice, mix, in a glazed earthenware basin resting on sand, 4 ozs. of water with 4 ozs. of H_2SO_4, and pour this over a mixture of 1 lb. of common salt and $\frac{1}{4}$ lb. manganese dioxide.

(*d*) **Oxygen**—(*e*) **Ozone**.—These must be nascent to have any germicidal action, and are outside the range of practical disinfecting.

3. LIQUID AGENTS.

(*a*) **Corrosive Sublimate** ($HgCl_2$).—This salt is soluble in 16 parts of cold water, or 3 of hot.

The most convenient way is to keep a saturated alcohol solution (25 *per cent.*), *to which a little ammonium chloride has been added.* This will keep indefinitely, and can be diluted with water as or when required.

Sea water is well suited for making perchloride solutions.

It is decomposed by lead, tin, copper, &c., and, therefore, should not be kept in metal receptacles.

To prevent accidents with the poisonous colourless solution it should be coloured with indigo or some other dye.

The germicidal action is probably due to chemical action on the mycoprotein of the germ.

Insoluble and inert substances are formed with albuminous matter; its *range of usefulness as a practical disinfectant is*, therefore, *very limited.*

1 in 1,000 for half an hour destroys non-sporing bacteria.
1 in 500 for one hour destroys spores.
1 in 2,000 for two hours *may* disinfect immersed articles.
1 in 15,000 will prevent putrefaction and fermentation.

(*b*) **Carbolic Acid** (C_6H_5OH).—This is the chief constituent of the acid portion of coal-tar oil. The crystals are soluble in about 15 parts of cold water; therefore, a saturated solution is about 7 per cent.

Solutions of 2 to 5 per cent. are those commonly used, and will kill non-spore-bearing organisms.

It cannot be relied on to kill spores.

It is not destructive to fabrics in strengths used, and, moreover, does not actively coagulate albumen as does corrosive sublimate. It can, therefore, be used for the disinfection of soiled linen, excreta, &c., and has a fairly wide range of application.

(*c*) **The Cresols.**—*Tricresol* is a mixture of metacresol (liquid) with ortho- and para-cresols (solids).

It is three times as powerful, bulk for bulk, as is carbolic acid. A 1 per cent. solution is a practical disinfectant.

It is a prompt germicide, does not coagulate albumen, and will kill spores.

Jeyes' Fluid.—This is practically 10 *per cent. of cresols with a little carbolic acid held in solution by soap.* It is a dark-brown thick liquid, forming a white emulsion with water.

It is equal or superior to phenol, and can be used widely as a good household or quarantine disinfectant.

Lysol contains about 50 per cent. of cresols with neutral potash soap. It is a brown, oily liquid, mixing with water in all proportions, and forming a soapy liquid.

It is more powerful than carbolic acid, and ranks with tricresol as a germicide. It is an admirable laboratory disinfectant for cleaning the hands, sterilising slides, &c.

(*d*) **Formalin** ($CHOH$).—A solution (40 per cent.) of formaldehyde gas in water.

The remarks made on the latter under the head of gaseous disinfectants will equally apply to the liquid.

It is *probably the most valuable disinfectant we possess.*

4 per cent. solution is equal to 1 in 1,000 $HgCl_2$.

4 per cent. solution is superior to 5 per cent. carbolic.

Formalin is a true deodorant. One smell is not masked with another, but odourless and sterile new compounds are formed with albumen.

1 in 25,000 to 1 in 50,000 will inhibit the growth and development of bacteria: 1 to 4 per cent. will kill them in a very short time. A minute trace added to wine or milk preserves them for a lengthened time.

(*e*) **Potassium Permanganate** ($KMnO_4$).—Dark-purple crystals soluble in 16 parts of cold and 2 parts of boiling water.

(Stains can be removed by oxalic acid, hydrochloric acid, or lemon juice.)

It readily gives up oxygen to organic matter, and, therefore, is quite useless as a disinfectant in the presence of much organic matter. Apart from this, it is a germicide of considerable power.

1 in 833 will kill pus cocci in two hours.

5 per cent. solution will kill spores in one day (*Koch*).

Its application is chiefly in the disinfection of contaminated tanks and wells. Enough should be added to give the water a tinge of colour.

Toxicologically, 8 to 10 grains have been taken without injury in dilute solution.

(*f*) **Lime Solutions.**—These are, up to a certain point, both efficient and cheap. Quicklime will destroy both organic matter and germs. Slaked lime is made by adding a pint of water to 2 lbs. of lime.

Whitewash ($Ca(OH)_2Aq$) is useful for disinfecting walls, &c. It should be freely applied to stables, outhouses, and other build-

ings after infection. It is also useful for mixing with excreta. Cholera germs are destroyed in a few hours by a 2 per cent. solution of crude lime.

Chloride of Lime (CaClOCl), commonly called bleaching powder.—It ranks with unslaked lime as a germicide. It is prepared by passing nascent chlorine over calcium hydroxide.

Only a 1 per cent. solution can be made with water. It bleaches and destroys fabrics.

Its chief use is for scrubbing floors and woodwork, and disinfecting excreta.

(*g*) **Ferrous Sulphate** ($FeSO_4$).—It is a good deodorant. Its germicidal power is very feeble. A 5 per cent. solution will scarcely kill typhoid bacilli in three days.

In the French army a 10 per cent. solution is much used for disinfecting latrines.

(*h*) **Zinc Chloride** (ZnCl) was at one time valued, and is still used. It is deliquescent, and very soluble in water.

In 1 in 500 solutions it is antiseptic—*i.e.*, will inhibit the growth of bacteria.

(*i*) **Soap Solutions.**—These are not very markedly disinfectant, but, with hot water and mechanical cleansing, they have a wide range of usefulness.

Medicated Soaps are, as a rule, poor. Carbolic acid and corrosive sublimate can combine with the soap, and, therefore, their disinfecting value is diminished. There is very little disinfectant in the soap. Very little soap is used, and that is largely diluted, so that the ultimate disinfecting action is practically *nil*. McClintock's soap, containing bin-iodide of mercury, is said not to combine with the disinfectant, not to attack metal, not to coagulate albumen, and is a good germicide.

II. APPLICATION OF DISINFECTANTS.

Air.—One of the gaseous agents should be employed.

Bandages.—Boiling, steaming, or dry heat.

Bed Linen.—Boiling, steaming, or immersion. (Wool will shrink in boiling.) 5 per cent. formalin for two hours, then remove and wash.

Beds.—Hot carbolic.

Bedding.—Pressure steam.

Brushes.—If no glued backs, boil or steam: if glued backs, cleanse in soap, wash, and immerse in 5 per cent. formalin.

Books.—These cannot be disinfected on shelves. The exposed surface, however, is the only part probably infected if the book has not been opened.

They should be stood open on wire trays in a closed chamber and exposed for twelve hours to formalin vapour at a temperature of 80° C.

The binding, illustrations, and print are not injured.

Cadavers.—The corpse at death should be wrapped in a sheet saturated with strong disinfectant if death has been due to infectious diseases.

If subsequent cremation is not feasible, the body should be surrounded with quicklime and the coffin tightly sealed.

Carriages.—These may be run into a closed shed and subjected to the vapour of formalin or SO_2, which will give surface disinfection.

If the vehicle has been used for infectious disease, all loose upholstery should be removed and separately dealt with; the rest of the interior being saturated with a strong solution of formalin, the woodwork scrubbed with lysol, and the whole then aired in the sun.

Clothing.—This may be exposed to steam, dry heat, gases, or solutions.

Leather, hide, skins, fur, or woollen goods should not be boiled or steamed.

Formalin gas admitted to a chamber with a partial vacuum is an admirable means of treating clothing.

Steam is excellent for many articles, especially if kept dry by high surrounding temperatures. SO_2 is of little use owing to lack of penetration.

Colours.—Articles with colours should not be subjected to steam, boiling water, SO_2, or chlorine. Solutions are also liable to make them run. They should be treated with formaldehyde gas.

Carpets.—Should be first exposed to disinfecting gas, such as formalin.

Stains of excreta or discharges should be scrubbed with hot solution of lysol. The carpet can then be steamed and finally aired in the sun.

Curtains.—Steam.

Excreta.—Cover from flies, &c. Mix thoroughly with milk of lime (1 of quicklime to 8 of water).

Or formalin can be incorporated, acting both as germicide and deodorant. When possible, incinerate.

Food.—In districts of cholera, typhoid, or dysentery, all salads, celery, tomatoes, fruit, &c., should be immersed for half an hour in 3 per cent. tartaric acid, and afterwards washed in boiled water. Surface disinfection of fruits and vegetables may also be effected by immersion in 5 per cent. solution of formalin which does not harm them, and is non-poisonous.

Floors.—Soak in 5 per cent. carbolic. Then scrub with soap and hot water.

Furniture.—If this has not come in contact with patient or infectious material, gaseous disinfection of the room with formaldehyde will be sufficient.

If contamination has occurred, any upholstery should be removed and burned; and the woodwork washed with strong formalin.

Glass.—This, as well as china, porcelain, &c., should be boiled.

Hands.—A nail-brush should be used with hot water and 5 per

cent. of lysol. This is sufficient for any or every circumstance if carefully carried out.

Hats.—Formaldehyde gas.

Houses.—Treat each room separately with formaldehyde fumigations. Subsequently, wash throughout with hot water and soap, air thoroughly, re-paper, paint, and whitewash throughout.

Instruments (Schimmelbush's method).—Boil for fifteen minutes in a 1 per cent. solution of sodium bicarbonate. It does not rust or affect the cutting edge.

Leather.—This, together with skins, fur, &c., are ruined by boiling, steaming, or immersing in strong solutions. Dry formaldehyde gas is probably the best method.

Mails.—Letters probably have, as a rule, but little connection with the spread of plague, cholera, typhoid, or tuberculosis.

They are a *grave source of danger in smallpox* cases, although exempted from sanitary treatment by the various sanitary conventions. They also probably carry measles and scarlet fever.

The best disinfection is to clip a tiny corner of the envelope, drop in two or three drops of formalin. Spray formalin inside the bag, and a fine spray between the letters as they are re-inserted. Then tightly close the bag. By the time it reaches its destination it should be quite safe.

Milk.—Either boil or pasteurise. The latter method is carried out by heating to 75° C. (180° F.) for half an hour, then keep on ice. This does not coagulate the casein.

Money.—Coins should be boiled for half an hour.

Paper money should be sprinkled with formalin and shut in a warm box for six hours.

Paintings.—Uninjured by formaldehyde, which should always be used.

Rags.—Burn, or expose to steam under pressure.

Rubber.—Injured by dry heat. Immerse in formalin solution.

Silk.—Injured by steam. Use formaldehyde gas.

Sputum.—Expectorate (in infectious diseases, such as phthisis, diphtheria, &c.) in covered cuspidors containing 2 per cent. lysol.

The sputum should subsequently be burned, as also soiled handkerchiefs, &c.

Stables.—Remove loose articles for treatment separately. Hay and straw should be removed and burnt. Spray throughout with strong formalin, and subsequently whitewash.

Tableware.—Boil.

Tents.—Spread in sun and soak in formalin.

Urine.—Add 3 to 5 per cent. of formalin.

Vessels.—Shipboard disinfection will vary with the disease at stake.

If for plague, disinfect all holds, cabins, stores, &c., with SO_2 gas under pressure for some hours, keep shut up for twenty-four hours to destroy vermin, &c. Then open, wash with Jeyes' fluid, burn rats, paint, and whitewash throughout.

If for smallpox, much the same procedure, but chief care directed to the actual domicile of the patient.

If for cholera, the same procedure throughout the ship, though less gas will be needed. The bilges should also be pumped out and Jeye's fluid inserted.

The water tanks should be emptied, and steamed from the ship's boilers. The stores also should be overhauled, and any vegetables or suspicious fruits, &c., condemned.

Care should, in all cases, be taken to enquire into the nature of the cargo and ballast which can be dealt with as circumstances may demand.

If dry, SO_2 will practically damage no cargo.

Cabins should be tightly sealed and formaldehyde generated by Walker's method (*q.v.*).

Walls.—Scrub with hot lysol solution or strong formalin solution. Re-paper and whitewash thereafter.

Water.—Filter through Pasteur-Chamberland or Berkefeld filters, and then boil.

Wells.—Do not trust to potassium permanganate if well is infected. Either add some and boil all water before use (if the well cannot be emptied or no other supply is available); or, if the well can be emptied, add 1 per cent. formalin, scrub, allow to stand, and then pump out.

Or quicklime may be used in the same way, half a barrel being stirred up.

Wool.—Formaldehyde gas.

CHAPTER XLII.

THE BLOOD.

MORPHOLOGY—ENUMERATION OF CORPUSCLES—PREPARATION AND STAINING OF FILMS.

Morphology of the Blood.

I. Red Corpuscles (*Erythrocytes*).—Biconcave, non-nucleated, circular discs. Yellow colour due to hæmoglobin. In health all cells contain same amount of hæmoglobin. They have no cell membrane. They have no contractile protoplasm. The average diameter is 7·5 μ. Average number in man is 5,000,000 per c.mm. Average number in woman is 4,500,000 per c.mm. Number is relatively high for two or three days after birth. Number is increased after fasting. Number is decreased—(1) After food; (2) after prolonged fatigue; (3) during menstruation; (4) during pregnancy.

Pathological Alterations—1. **Polycythemia** (increase in number).—Relatively infrequent. Occurs during fasting. Occurs in cholera and diarrhœa. May reach 6,500,000 to 7,000,000 per c.mm.

2. **Oligocythemia** (diminution in number).—Of frequent occurrence. Occurs after hæmorrhages. Occurs in anæmic conditions. Is serious when number approaches one million. Is fatal below half a million (*Hayem*).

3. **Macrocythemia** (increase in diameter).—Found with severe anæmias. May reach 14 μ in diameter.

4. **Microcythemia** (diminished diameter).—Commonly found in chlorosis. May occur in lymphatic leukæmia. May occur in Hodgkin's disease. Size may diminish to 3 μ in diameter.

5. **Poikilocytosis** (altered shape). — May become oval, pear-shaped, or elongated. Found in most cases of anæmia and leukæmia. Very pronounced in pernicious anæmia.

6. **Chlorocytosis** (diminished hæmoglobin).—Corpuscles partially or totally decolourised. Occurs in adynamic typhoid. Occurs in hæmorrhagic smallpox. Found occasionally in Hodgkin's disease. Found also in chlorosis.

7. **Chromatophilia** (elective staining reaction).—In circulating, living, blood-red corpuscles do not stain (*achromatophilia*). When dead and fixed they only take the acid stain in a mixture of aniline colours. This is the normal reaction (*monochromatophilia*).

In pathological conditions—*e.g.*, anæmia, measles, scarlet fever, smallpox, typhus, &c.—they may lose their elective property and stain with several colours (*polychromatophilia*).

8. **Nuclecoythemia** (nucleated red corpuscles).—Present in intrauterine life and certain pathological conditions. Generally of two sizes.

(*a*) *Normoblasts.*—Diameter as in ordinary corpuscle (7·5 μ). Nucleus usually single and round (or oval). Smaller than the nucleus of a lymphocyte. Usually eccentrically situated. Stains very deeply. Found in primary and secondary anæmias and splenomedullary leukæmia. Indicates a severe anæmia, but one in which an effort is being made to regenerate (*Coles*).

(*b*) *Megaloblasts.*—Diameter 10 to 15 μ. Nucleus may occupy two-thirds of the cell. Stains less deeply than normoblast. Protoplasm stains deeply with eosin. Occur in fœtal red medulla. Found in pernicious anæmias. Are signs of perversion of hæmogenetic action of the marrow. Indicate retrogression of a degenerative nature (*Coles*).

II. White Corpuscles (*Leucocytes*).—Spherical masses of protoplasm. Contain no hæmoglobin. Undergo amœboid movement. Some of them are phagocytic. Vary in size from 6 to 11 μ. Average number 6,000 to 8,000 per c.mm. Proportion of white to red 1 to 500. Number increased during first few days of life.

Increase of 18 to 20 per cent. after a meal. Increase more pronounced after proteid than mixed diet. No appreciable difference during fasting. Slight increase during pregnancy. Increase is called **Leucocytosis**. Decrease is called **Leucolysis**. Leucolysis does not occur as a physiological condition, except to a slight extent during fasting.

Proportion of Leucocytes in Normal Blood.

Polymorphonuclear, . . .	70 per cent
Lymphocytes, ...	25 ,,
Large mononuclear, . . .	4 ,,
Eosinophiles, ...	1 ,,
	100 ,,

Differentiation of Leucocytes—**Lymphocytes.** A normal constituent of blood. Smallest of all leucocytes. Size equal to or less than red corpuscle. Diameter 6 to 7·5 μ. Single large round nucleus occupying nearly whole cell. Stain more deeply and more quickly than other leucocytes. *Constitute 25 per cent. of leucocytes in blood.* Number increased—(1) after food; (2) in lymphatic leukocythemia. Number diminished—(1) during starvation; (2) lymphatic gland disease. Originate from lymphatic tissue. Probably are immature forms of large mononuclear. Neither amœboid nor phagocytic.

Large Mononuclear Leucocytes.—Normal constituents of

blood. Measure 8 to 12 μ in diameter. Protoplasm contains no granules. Protoplasm stains slightly with *basic* aniline dyes. Nucleus is large, round (or oval), and occasionally kidney-shaped. Nucleus stains faintly. Actively amœboid and phagocytic. *Constitute* 4 *per cent. of leucocytes in blood.* Number increased in cachectic states.

Polymorphonuclear Leucocytes. — Normal constituents of blood. Measure 7·5 to 9·5 μ in diameter. Protoplasm contains fine granules. The granules stain with acid aniline dyes. Stained with eosine and hæmatoxylin, the protoplasm is uniformly pink, and no granules can be made out with a $\frac{1}{12}$ objective. Nuclei are 3 to 6 in number, and round or oval in shape, connected by thin threads of chromatin. Nuclei stain deeply. The corpuscles are actively amœboid. They are also phagocytic. Pus consists mostly of polymorphonuclear cells. They *constitute* 70 *per cent. of the leucocytes in normal blood.* Number is increased in acute inflammatory diseases, leukæmia, secondary anæmia, &c. Number is diminished in long-continued non-inflammatory fevers, in pernicious anæmia, &c.

Eosinophile Cells.—Normal constituent of blood. In size are between large mononuclear and polymorphonuclear. Diameter, 10 to 11 μ. Protoplasm has highly refractile granules. Granules have great affinity for acid aniline dyes, especially eosine. Nucleus may be single, double, or indented. Generally horse-shoe shaped. Nucleus usually eccentric, and does not stain as deeply as polymorphonuclear. They are amœboid. They are *not* phagocytic. *Usually constitute* 1 *per cent. of leucocytes in blood.* They may reach 11 per cent. in healthy blood. Relatively numerous up to 13 or 14 years of age. Number is increased in asthma, skin diseases, and helminthiasis. Number is diminished in croupous pneumonia, when they may even be absent.

Myelocytes (marrow cells).—*Never present in normal blood.* Occur in bone marrow. May reach size of 20 μ. Protoplasm contains fine granules. Granules are basophile. Nucleus is single and often indented or lobed. Nucleus stains only faintly. Occur in blood in small numbers in chronic cachexias, anæmias, syphilis, &c. Occur in enormous numbers in spleno-medullary leukæmia.

Mast Cells (granular basophiles).—*Never present in normal blood.* Found in bone marrow and connective tissue. Often exceed 20 μ in diameter. Protoplasm has dense granules obscuring nucleus. Granules stain deep purple-blue with dahlia and methylene blue. Unstained granules not very refractile. Nucleus stains very faintly, and appears as a cavity. They are non-amœboid and non-phagocytic. They are very rare in the blood. Have been found in leukæmia and in the reaction stage of cholera.

III. Blood Plates (*Hæmatoblasts*).—Third corpuscular elements of blood. Discovered by Hayem. Round or oval bodies. Diameter 2 to $3\frac{1}{2}$ μ. Faintly yellow and slightly granular. Remarkably adhesive, quickly clustering together, and becoming spinous. Stain faintly with aniline dyes, having an amphophile reaction.

Number, 250,000 per c.mm. Function is formation of white blood clot.

Number increased in—(1) non-febrile anæmias; (2) hæmorrhages; and (3) leucocythemia.

Number diminished in—(1) infectious fevers; (2) starvation; and (3) advanced cachexia.

Number unaltered in tuberculosis and pneumonia.

Enumeration of Blood-Corpuscles.

Gower's Hæmocytometer is the one in more general use in England, but is somewhat complicated and costly. In this, 995 c.mm. of saline solution are measured and put in the mixing jar. 5 c.mm. of blood are then drawn into a capillary pipette and blown into the diluting fluid, which is then mixed with a glass stirrer. A drop of this diluted mixture is placed in the counting cell, and covered with a cover-glass. Each square measures $\frac{1}{500}$ c.mm., and the dilution is 1 in 200. Therefore, the calculation is—

$$\frac{\left\{\begin{array}{l}\text{No. of corpuscles}\\ \text{counted} \times 200 \times 500\end{array}\right\}}{\text{No. of squares counted}} = \begin{array}{c}\text{No. of corpuscles per}\\ \text{1 c.mm of blood.}\end{array}$$

The **Thoma-Zeiss Hæmocytometer** is a more convenient and accurate instrument. A pipette which can be carried in a small case not much larger than that of a clinical thermometer and a small bottle containing about 30 drops of diluting fluid are the only necessary impedimenta.

The blood is drawn into the pipette (Fig. 86) up to the mark 1·0. The end of the pipette is then wiped and immersed in the diluting fluid, which is carefully drawn in until the whole reaches the mark 101. The dilution is thus 1 in 100, and the whole can be kept until some convenient time for examination.

On the examining slide the depth of the cavity is 0·1 mm.

The 16 large squares cover an area of 1 sq. mm. Each of these squares is divided into 25 smaller squares. Hence, each of the smaller squares contains $\frac{1}{10} \times \frac{1}{16} \times \frac{1}{25}$ c.mm. ($\frac{1}{4000}$ c.mm.). The calculation, therefore, will be—

Fig. 86. Pipette.

$$\frac{\text{No. of corpuscles counted} \times 100 \times 4{,}000}{\text{No. of small squares counted}} = \begin{array}{c}\text{No. of corpuscles} \\ \text{per c.mm.}\end{array}$$

Blood Examination.

Examination of the blood is often of such extreme importance that a thorough knowledge of technique is indispensable to the tropical practitioner or worker.

Normal as well as abnormal blood should be readily recognised.

The number, proportion, and characters of blood cells have been already dealt with. It now remains to consider the methods of practical blood examination.

The best time for the beginner to examine malarial blood is just at the end of the apyrexia (*Brit. Med. Journ.*, 19th October, 1895) when the parasites are pigmented and large.

It is often necessary to observe the blood while still alive and fresh. Only thus can certain vital changes be observed, such as movements of filariæ, trypanosomes, &c., developmental stages of parasite growth, amœboid and phagocytic properties of leucocytes. We can, therefore, divide the subject into two parts.

I. Examination of Fresh Blood—1. *Coverslip Method.*—The method is to obtain a drop of blood on a coverslip. This may

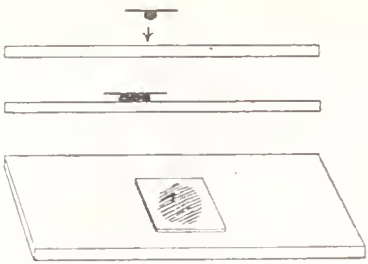


Fig. 87.—Coverslip.

be got from the finger or the lobule of the ear. The latter is the more convenient, cleaner, and less painful place. It is first cleaned with a little alcohol, and wiped dry with a clean rag. A puncture is then made with a needle (either an ordinary or a surgical one), sterilised by passing it through the flame of a spirit lamp, or, at a pinch, a burning match.

A small drop of blood is squeezed out and taken up on the centre of a clean coverslip (Fig. 87). This is dropped at once on to a slide, when the live blood spreads out and can be examined.

In a well-made film by this method, three zones are apparent. In the centre there is mainly serum with a few scattered corpuscles. At the outer edge the film is thick and irregular, with the corpuscles in rouleaux.

Preparation of Films.

The intermediate area, which is the best for the examination of red corpuscles and parasites, the corpuscles will be found fairly plentiful and flat, and not to any extent overlapping.

To get good films the slides and coverglasses must :—

a. Be free from grease.

b. Be free from grit ; and

c. They should both be *slightly* warmed just before the operation.

d. The drop of blood should be sufficiently small not to reach the edges of the coverglass when it has spread.

2. *Braddon's Method.*—A drop of blood is obtained in the same way as before. A coverglass free from grit and grease is placed on

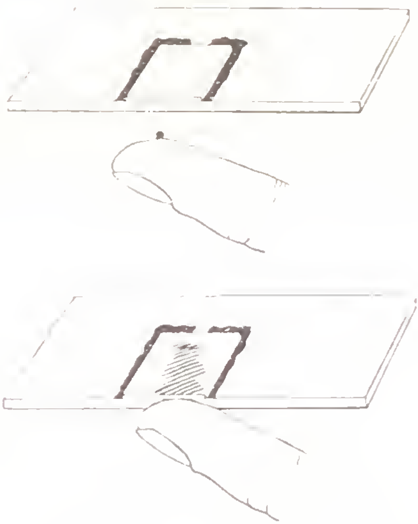


Fig. 88.—Making a blood film.

an equally clean slide, so that its edge corresponds with the edge of the slide.

Vaseline is then applied to the sides of the coverslip and part of the posterior, but not to the edge corresponding to that of the slide (Fig. 88).

If the edge of the slide be applied to the drop of blood, the latter will spread by capillary attraction and give a film good enough for examination.

The slides can be prepared at home or in the laboratory, and taken in a box for use.

Vaseline is, however, a messy thing. The coverglass may slip with a shake or jar, the vaseline may get on the objectives. A modification may, therefore, be used. The following is the author's

practice :—The coverglass having been applied to the slide as before, a glass rod (or even a penholder) may be heated in a lamp flame and rubbed on a cake of hard paraffin, and immediately applied to the sides of the coverslip (Fig. 89).

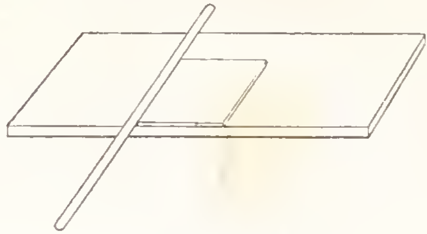


Fig. 89.—Fixing coverslip.

If this operation is performed a couple of times on both *sides* of the coverslip, it will act as a cement which is dry in a second. The coverglass cannot slip. The microscope objectives cannot be covered with vaseline, and no application is even necessary on the third side of the coverslip.

If it is required to *stain the fresh blood*, Braddon's solution may be used :—Pot. cit. 1, meth. blue ½ to 2, water 100.

To use this by method 1, a drop of the stain is placed on the slide, and the coverslip with the blood is dropped on to it so that they mix.

To use it by method 2, a drop or two of the stain is placed on the ear or finger, and the needle puncture is made through this drop, so that the exuding blood may mix with the stain.

Living red corpuscles do not take the stain, but the disadvantage of the method is that the water in the applied stain liberates the hæmoglobin, and the dissolved hæmoglobin precipitates the stain, or debris is stained by the stain. This occasionally gives rise to a complicated stained arrangement in the interior of the red corpuscles which may be mistaken for parasites.

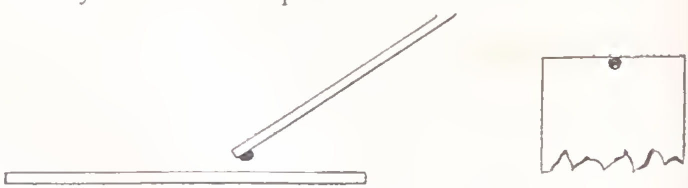


Fig. 90.

II. Examination of Dried Films.—This is a most useful way of examining blood, as the films can be stored or transmitted for subsequent examination either before or after staining.

To get good films it is essential that the skin should not be touched when obtaining the drop of blood, and also that the slide should be clean and free from grease.

Method 1.—Take up a drop of blood on the edge of a microscope slide, and bring the drop in contact with another slide near its end (Fig. 90).

Wait until the drop has spread across the slide (Fig. 91), and then push the one along the other (Fig. 92).

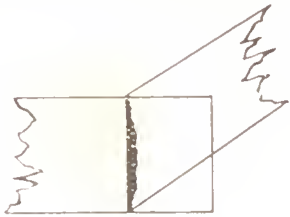


Fig. 91.

The thickness of the blood film can be varied by changing the angle between the two slides.

An acute angle will give a thin film; an obtuse, a thick one.

For examination for the malarial parasite, an angle of 45° is about right (Fig. 92).

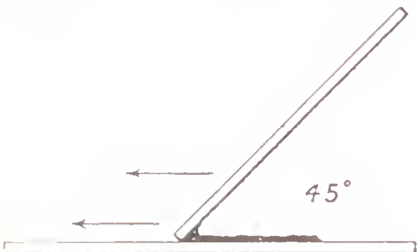


Fig. 92.

Method 2.—A drop of blood is taken on a slide, near one end (Fig. 93).

A shaft of a straight needle is then applied to it, till the blood spreads by capillary attraction along the shaft of the needle (Fig. 94).

Fig. 93.

The needle is then drawn along the slide (Fig. 95), and a good film should be obtained.

Method 3.—The end of a strip of cigarette paper is brought in contact with the drop of blood, and is then applied to the end of a

slide. The blood spreads out, and by pulling the free end right along the slide, a somewhat indifferent though usually workable film will be left.

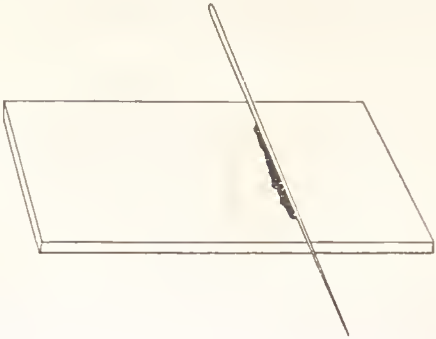


Fig. 94.

Films made in any of these ways should not be heated, but should be dried quickly in the air by movement.

This completed, it should be fixed, or else water manipulations would dissolve the hæmoglobin and wash off the film.

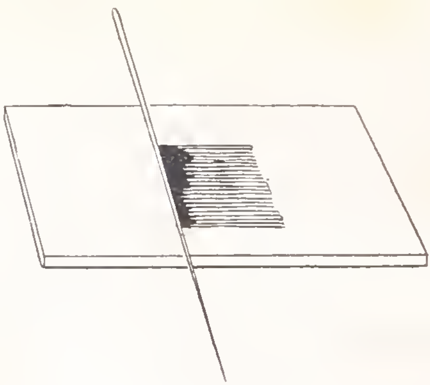


Fig. 95.

Fixation of Films.—There are various methods, *e.g.* :—

1. With Leishman's staining no previous fixation is necessary.
2. Immerse in alcohol five or ten minutes, and then dry in air.
3. Immerse in alcohol 1, ether 1, for five minutes, and then dry in air.
4. Expose to vapour of formalin for two minutes.

Films thus fixed, if kept in a dry place, can be stained at leisure.

Staining of Films.—A general consideration of stains will be found in Chapter xxxix. of this volume.

I therefore give, here, only the technique of the two best working stains for blood work.

1. EOSINE AND HÆMATOXYLIN give excellent and reliable preparations for blood examination. The following is the process adopted by Coles:—

a. Films are dried and fixed as mentioned.

b. They are then stained in eosine (0·5 per cent. sol. in [50 per cent.] alcohol) for half a minute, time not important.

c. Washed in water, and, while wet,

d. Stained in hæmatoxylin half to ten minutes (filtering the solution before use).

Hæmatoxylin, ...	1 gr.
Alum, ...	10 grs.
Distilled water, ...	100 c.c.
Alcohol, ...	5 c.c.
Camphor, ...	small piece.

M.

(This solution can be used after two months, and can be used for some years. Dilute with an equal volume of water for use.)

e. Washed well with filtered water. Dried in air. Cleared with xylol. Mounted in balsam.

The advantages of this method are:—

(*a*) Equality of stain intensity.
(*b*) Red corpuscles brightly stained with eosine.
(*c*) Do not fade with prolonged washing.
(*d*) No granules of stain or dirt.
(*e*) Staining time not necessarily exact.
(*f*) Equally excellent for all pathological conditions.

2. LEISHMAN'S STAIN.—Dr. Leishman prepares his stain as follows:—

(*a*) A 1 per cent. solution of Grubler's methylene blue in distilled water, is made alkaline by 0·5 per cent. of sod. carbonate. The solution is then heated to 65° C. for twelve hours, and then allowed to stand for ten days.

(*b*) A 1 in 1000 solution of Grubler's eosine (Extra B) is made.

Equal volumes of these two solutions are mixed in an open vessel and allowed to stand six to twelve hours with occasional stirring.

The precipitate is collected on a filter, washed with distilled water, and the insoluble residue dried and powdered.

The stain in this dried form can be obtained from Grubler or his English agents, also from Burroughs & Wellcome.

For use.—0·15 grm. of the stain is dissolved in 100 c.c. of pure methyl alcohol.

Method of Staining.—Films of blood, or smears from marrow,

spleen, buboes, &c., are made in the usual way on perfectly clean coverglasses or slides, and allowed to dry in the air.

No fixation is necessary.

The film is covered with a few drops of the stain, and rotated gently for one minute.

Double the quantity of distilled water is then added, and intimate mixture hastened by rotation. The diluted stain is allowed to remain for five minutes, after which it is gently washed off with distilled water, and left in water for one minute. It is then dried and mounted.

The process is simplicity itself, for the whole operation can be completed in seven or eight minutes; and no reagents are necessary beyond a few drops of the stain and a little distilled water.

The part of the procedure in which the film is allowed to soak in distilled water for a minute after staining and washing, has a triple importance.

It intensifies the Romanowsky staining.

It removes the remains of the deposit.

It turns the red corpuscles pink.

Appearances in Successful Films Stained with Leishman's Stain.—1. Red corpuscles. Transparent pink.

2. Polymorphonuclear leucocytes. Nuclei purple-red.

3. Large mononuclear leucocytes. Nuclei ruby-red. Protoplasm pale blue.

4. Lymphocytes. Nuclei ruby-red (more deeply stained).

5. Eosinophile cells. Nuclei ruby-red. Granules pale pink.

6. Mast cells. Nucleus red. Granules purple-black.

7. Nucleated red corpuscles. Nucleus black. Protoplasm grey.

8. Bacilli, &c., blue.

9. Malarial parasites, blue, with ruby-red chromatin.

10. Trypanosomes, purple-red, with red nucleus, centrosome, and flagellum.

To find Crescents. — *Manson* advocates — 1. A thick film taken and dried.

2. Dipped in water one or two seconds.

3. Fixed by alcohol on the wet slide.

4. Then stained.

Better alternative method is (*Coles*)—1. Thick film dried.

2. Fixed in alcohol.

3. Immersed in acetic acid (2 drops to a watchglass of water).

4. Washed. Stained and mounted.

To Stain Flagellated Body.—*Manson* recommends (*Brit. Med. Journ.*, 10th July, 1897)—1. Oblong hole, $1 \times \frac{3}{4}$ inch cut in centre of *thick* strip of blotting-paper 3×1 inch.

2. Strip moistened with water, and laid on slide.

3. Another slide is taken and breathed on: and a drop of crescent-positive blood spread on it.

4. Immediately placed, film downwards, over the blotting-paper cell.

5. After three-quarters of an hour, slide removed and dried over a spirit lamp.

6. Then fixed in absolute alcohol for five minutes.

7. A few drops of 10 per cent. acetic acid poured on to film, and left till hæmoglobin dissolved out.

8. Washed, dried, and stained.

9. Washed, dried, and mounted.

APPENDIX I.

INTERNATIONAL SANITARY CONVENTIONS,

Being a RÉSUMÉ *gleaned from a* TREATISE ON PLAGUE,
by PROF. W. J. R. SIMPSON,
M.D., F.R.C.P., D.P.H.

"WITH the disappearance of plague from Egypt and Turkey in the middle of the nineteenth century, the same urgency for precaution against the spread of plague no longer continued. The lazarettos, quarantines, and cordons sanitaires were now used to meet the invasion of cholera. It was considered advisable that in times of danger, representatives of the European Powers should meet and discuss the means of defence which might be adopted in common, for frontier and for seaport."

The following are the chief International Conventions:—

1st Convention at Paris, 1852.
2nd ,, ,, *Constantinople*, 1866.
3rd ,, ,, *Vienna*, 1874.

Here the guiding principle of action was changed, the state of health of those on board a ship being considered, rather than the fact that it had arrived from an infected port.

The period of incubation became the limit of detention.

4th Convention at Rome in 1885.

At this it was determined that quarantines and cordons for cholera were useless.

5th Convention at Venice, 1892.
6th ,, ,, *Dresden*, 1893.
7th ,, ,, *Paris*, 1894.

These three conventions had reference to cholera. No special points of note occurred.

In the latter year the bacillus of plague was isolated for the first time, and with the occurrence of a plague pandemic, international attention was forcibly drawn to the matter, resulting in two important conventions.

"In *1897*" the *8th International Convention* "of the European Powers was held *at Venice*, and an agreement was signed, in which it was agreed that certain protective measures, having for their object efficiency, but at the same time the avoidance of unnecessary restrictions on commerce, should be put into force against the threatened invasion of plague from the East." Infringement by any one of the signatories might result in stringent measures being enforced.

The idea at the time was, that the spread of plague was associated with sick persons and their effects, and that the detention of contacts should be based on the period of incubation.

The chief regulations were as follows:—

1. International notification of the occurrence of plague.

2. Medical inspection of crew and passengers leaving infected ports.

3. Special precautions as to such ships when passing through the Red Sea or Persian Gulf.

4. Special precautions *re* pilgrims from an infected country.

5. Procedure *re* vessels arriving from an infected port.

Such vessels are *Healthy*, if they have left the infected port for ten days and had no plague.

They are considered *suspected* if they have had plague on board, but no cases during the previous twelve days.

If cases have occurred within that time, they are termed *infected.*

All ships from infected ports are subject to medical inspection on arrival. "Healthy" ships are to be given free pratique on arrival, but passengers and crew have to submit to surveillance for ten days from the date of leaving the infected port.

If the ship is a "suspected" one, disinfection of ship and baggage, &c., is undertaken as well as the observation of crew and passengers in their own homes.

With an "infected" ship, the sick are landed and isolated. The crew and passengers are liable to be isolated at a quarantine station, or on board till they have obtained free pratique. Thorough disinfection of ship and contents is required.

6. Disinfection of merchandise and baggage is now to be carried out instead of the old system of quarantine. The importation of susceptible goods may be either prohibited, or the goods subjected to disinfection at the option of the government concerned.

The definition of such susceptible goods liable to be prohibited is :—

(*a*) Used linen, clothing, personal effects, and bedding.
(*b*) Rags, including those carried in bales as merchandise.
(*c*) Old sacking, carpets, and embroidery.
(*d*) Raw, untanned, and fresh hides.
(*e*) Animal refuse, horse hair, silk, and wool.
(*f*) Human hair.

7. Quarantine on land frontiers for travellers and merchandise is abolished.

8. With the exception of parcels, there is no disinfection of, or restrictions as to, mails.

9. Medical inspection at railways, custom houses, and special stations, and the surveillance of travellers from an infected area, are the measures which should be adopted.

10. Gipsies, vagabonds, emigrants, and other large bodies of people are subject to special measures at the option of each government.

Note.—This convention was not signed by Portugal, Turkey, Greece, or Servia.

The *9th International Convention was held in Paris in* 1903.

Since the Venice Convention there had been small outbreaks of plague in Oporto in 1899, in Glasgow in 1900, in Naples in 1901, and in Marseilles in 1903, and in none of these cases was the infection traceable to human imported cases.

Much, moreover, had been learned during the six years about the disease.

For instance, that rats are susceptible, and that there is a connection between rat plague and human plague; that infected

animals, clothing, and other material, as well as infected human beings, are means of transporting plague.

Acting on this knowledge, the regulations of this Paris Convention of 1903 *confirm those of the Venice Convention, except in two important respects*:—

(*a*) The period of detention of infected ships is reduced from ten to five days.

(*b*) In addition to disinfection, all rats must be destroyed.

The creation of an international sanitary office at Paris, to receive and transmit sanitary information, was agreed to as desirable.

Local measures on the part of the signatories usually devolve on municipal or government sanitary authorities.

Any additional measures may be taken as long as they do not run counter to the principles of the conventions.

Arrangements are accordingly made to provide for—

(*a*) Medical inspection of ships from infected ports.

(*b*) Isolation hospitals for incoming cases.

(*c*) Segregation buildings or camps for observation purposes.

(*d*) Surveillance ashore.

(*e*) Disinfection of suspected and infected ships, linen and luggage.

(*f*) Destruction of rats on ships from infected ports.

(*g*) Prevention of infected or suspected ships from mooring at wharves before rats have been destroyed.

(*h*) Inspection of fruit, grain, and other cargo from infected centres, to prevent the admission of rats.

Until recently the destruction of rats on ships was a difficult and costly process. The matter is now solved by the Clayton process of fumigation with SO_2, which gas has, moreover, the advantage of germicidal and insecticidal properties.

APPENDIX II.

VEGETABLE POISONS IN THE TROPICS.

By H. M. Ridley, M.A., F.R.S., F.L.S.

VEGETABLE poisons are limited to comparatively few orders.

The following scheme will show at a glance the poison-bearing classes and orders :—

1. GROUP.—*Thallophyta.*

(*a*) CLASS.—*Fungi.*

ORDER :—Basidiomycetes.

2. GROUP.—*Phanerogamia.*

(*a*) CLASS.—*Monocotyledones.*

ORDERS :—Palmaceæ.
Gramineæ.
Liliaceæ.

(*b*) CLASS.—*Dicotyledones.*

1. SERIES.—*Monochlamydeæ.*
ORDER :—Urticaceæ.

2. SERIES.—*Thalamifloræ.*
ORDERS :—Ranunculaceæ.
Menispermaceæ.

3. SERIES.—*Disciforæ.*
ORDERS :—Rutaceæ.
Anacardiaceæ.
Euphorbiaceæ.

4. SERIES.—*Calycifloræ.*
ORDER :—Leguminosæ.

5. SERIES.—*Hypogynæ.*
ORDERS :—Solanaceæ.
Loganiaceæ.
Apocynaceæ.
Plumbaginaceæ.

6. SERIES.—*Epigynæ.*
ORDER :—Lobeliaceæ.

Of the above orders, almost all the poisonous vegetable products occur in the four orders :—

Loganiaceæ, Apocynaceæ, Euphorbiaceæ, and Leguminosæ.

For purposes of convenience, this paper is divided in four sections.

1. The *internal vegetable poisons* arranged in the alphabetical order of their popular names.

2. The *injection poisons* which must be introduced into the skin or blood rather than into the intestinal tract.

3. The *drug poisons;* that is to say, the poisonous products of certain tropical plants which in themselves are not sufficiently potent to cause harm; but, when extracted, form dangerous drugs, and can be procured on the market as such.

4. *Chemical analysis* for detection of alkaloids and vegetable poisons.

SECTION I.

Internal Vegetable Poisons.

ACOKANTHERA VENENATA.—Nat. Ord., Apocynaceæ. A native of East and South Africa. Native names—*Dutch*, "Giftboom"; *Swaheli*, "Mtchungu."

ACOKANTHERA SCHIMPERI.—Nat. Ord., Apocynaceæ. Allied to above. Is a native of Abyssinia. Native names—"waba," "wabei," "wabayo."

Both the above are used medicinally in heart disease and whooping-cough. A decoction of the roots is also used as an arrow poison.

The toxic agent is a glucoside named "ouabain." Its composition and action are but little known.

ACONITE (*Aconitum ferox*) in India; native name—bish or bikh. (*Aconitum fischeri*) in China. Nat. Ord., Ranunculaceæ. Strictly speaking, is a north temperate zone plant occurring in Europe, N. America, and Siberia.

The roots are imported into most tropical places. They are from 2 to 3 inches long, and from $\frac{1}{2}$ to $\frac{3}{4}$ inch thick at the upper extremity; tapering, wrinkled, and blackish brown; whitish on section; beset with numerous rootlets. The taste is acrid and produces a numbness of the tongue.

Analysis.—It contains an alkaloid "aconitina" obtained from the root by maceration with spirit and subsequent precipitation by ammonia. The formula is $C_{33}H_{43}NO_{12}$, and the alkaloid is combined with aconitic acid. It also contains other alkaloids, such as "pseudaconitina," $C_{36}H_{49}NO_{11}$, "aconina," $C_{26}H_{39}NO_{11}$, the latter being supposed to be identical with "narcotine." It also contains sugar, resin, and fatty matter.

Antidote.—Atropine gr. $\frac{1}{60}$ given hypodermically, and repeated in twenty minutes, if necessary.

AGARICUS.—See *Mushrooms*.

ANAMIRTA PANICULATA.—See *Cocculus indicus*.

BAMBOO.—Dendrocalamus, sp. Nat. Ord., Gramineæ. Malay term, "Bulu Bambu."

The fine hairs from the bamboo sheaths are used criminally, being put into curry or coffee. It appears to be the common practice to add black sand to the hairs, which probably serves to sink them and render them inconspicuous.

A profound mechanical irritation of the intestinal mucosa is caused by the ingestion of these hairs, and is frequently fatal.

Probably the best treatment would be olive-oil enemata and olive oil by the mouth, one ounce each half-hour for four doses. Followed in three hours by a dose of castor oil. Milk diet only for some days. If persistent, treatment as for dysentery should be tried.

BHANG.—See *Cannabis sativa*.

Buta-buta.—See *Cerbera odollam*.

Calabar bean, Physostigma venenosum. Nat. Ord., Leguminosæ. A native of Africa, and not found elsewhere in the tropics.

Oblong, reniform flat beans of a chocolate colour, without bitterness, acrimony, or aromatic flavour.

Used in Africa as an ordeal poison.

Principle is physostigmina, an alkaloid of the composition $C_{15}H_{21}N_3O_2$, called also eserine, occurs as colourless or pinkish crystals, slightly soluble in water, readily soluble in alcohol or dilute acids. The aqueous solution becomes red on the addition of potash. The alkaloid causes contraction of the pupil.

Antidote.—Atropia gr. $\frac{1}{60}$ given hypodermically, and repeated in twenty minutes, if necessary. In bad cases, chloral; and strychnine gr. $\frac{1}{60}$ hypodermically.

Calamus.—See *Rotan*.

Cannabis sativa (Indian hemp, Bhang, Ganja). Nat. Ord., Urticaceæ. Is a native of India.

The stalks and leaves of the Indian hemp yield a resinous brown gum.

Characters.—The tops of the plants consist of one or more alternate branches, bearing the remains of the flowers and smaller leaves, and a few ripe fruits (glued together with resin), which are several inches long; harsh, of a dusky green colour, and has a characteristic odour.

Analysis.—Cannabinine, a resin on which its properties depend, is developed only in warm countries.

Antidotes.—1. Emetic.

2. Hot brandy and water, and vegetable acids (lemon, vinegar, &c.).

3. Strychnine hypodermically.

4. Blister to nape of neck.

Cassava.—Manihot utilissima (Bitter cassava). Nat. Ord., Euphorbiaceæ. Native of Brazil, and cultivated there for food. Rarely used in the East Indies, where the sweet cassava replaces it (Tapioca).

It has a fleshy root up to 40 inches in length, which yields hydrocyanic acid, as do the Rosaceæ, nearly all the Leguminosæ, and many other plants. Unless heated, it is very poisonous.

Antidote.—Atropine gr. $\frac{1}{60}$ given hypodermically, and repeated in twenty minutes if necessary.

Castor oil.—Ricinus communis. Nat. Ord., Euphorbiaceæ. Malay term, "Minyak Jarak." Probably a native of Africa, now ubiquitous.

Characters.—Seeds $\frac{1}{2}$ inch long, oblong, brown, with silvery marking, and has a small white caruncle at one end.

Used to produce intestinal inflammation, with dysenteric stools. Seven seeds have proved fatal.

Often used by native prisoners to produce a dysentery, and thus get to hospital.

CERBERA ODOLLAM.—Nat. Ord., Apocynaceæ. Malay term, "butabuta."

Is a littoral tree, a native of the East Indies; now distributed along all tropical Asiatic coasts, where it grows wild.

The latex is very poisonous, and contains a principle known as cerberin, probably a glucoside.

The latex is occasionally used for poisoning, producing violent inflammation of the intestines.

When dropped on the conjunctiva it produces blindness.

The fruits are used as a counter-irritant for muscular rheumatism.

CHERAKA MERAH.—See *Plumbago rosea*.

CROTON (C. Tiglium).—Nat. Ord., Euphorbiaceæ. A native of India; now ubiquitous in the tropics.

Is a small tree, sometimes 20 feet in height.

Seeds about the size of peas, are flattened, suborbicular, hard, and dark brown, with a pungent taste.

Antidotes.—An emetic should be at once administered, and the stomach washed out with olive oil or milk, 4 fl. ozs. to the pint of water; mucilaginous fluids and opium or morphine should then be given to check the pain and enteritis.

Bismuth subnitrate in large doses may also prove useful.

COCCULUS INDICUS.—Anamirta paniculata. Nat. Ord., Menispermaceæ. Native of India, not found elsewhere.

It is a tall climbing shrub. The fruit is the part used, and is known as Cocculus indicus.

It is of about the size of a large pea, reniform, wrinkled, and dark blue in colour.

The pericarp contains two alkaloids, which have toxic properties, and the seeds contain a neutral principle (a glucoside) called *Picrotoxin*, $C_9H_{10}O_4$.

Picrotoxin is a white crystalline powder, with a bitter taste, and no odour. It is a powerful poison, causing vomiting and clonic and tonic convulsions.

In medicine, picrotoxin is used externally in skin diseases, and for destroying pediculi, while, as an internal remedy, it is one of the most powerful remedies known for checking the night sweats of phthisis. Dose, gr. $\frac{1}{10}$.

Antidotes.—Emetics, chloral, potassium bromide.

DATURA FASTUOSA.—Native name Kechubong. Nat. Ord., Solanaceæ. Originally native of East Indies, but now ubiquitous in tropics. Rest of genus is South American.

It is a herb with broad, finely hairy leaves and large tubular white or purple flowers The fruit is a capsule containing numerous small seeds yellowish-brown, earshaped or obscurely triangular.

The whole plant is poisonous, and decoctions of the leaves, fruits and flowers are used for drugging, which usually consists in adding it to tea or other beverage.

It is rarely fatal, but is probably the most commonly used drug for criminal poisoning in the East.

Externally it is used as an anodyne for sprains and rheumatism.

The toxic agent is an alkaloid Daturina, $C_{17}H_{23}NO_3$, allied to Belladonna.

DENDROCALAMUS.—See *Bamboo.*

DERRIS ELLIPTICA.—See *Tuba root.*

ERYTHROPHLŒUM GUINEENSE.—See *Sassy-bark.*

GANJA.—See *Cannabis sativa.*

GLORIOSA SUPERBA.—Nat. Ord., Liliaceæ. Allied to Colchicum. Probably a native of India. Found also in tropical Africa, Malaya, &c. Of no economic use. A climbing plant with red and yellow flowers, often grown for ornament. Toxic properties not known. The rhizomes produce fatal irritation of the intestines. Cases of death have occurred from accidental eating.

GLUTA RENGHAS.—See *Rengas.*

HIPPOMANE MANCINELLA.—See *Manchineel.*

HYOSCYAMUS MUTICA — Henbane. — Nat. Ord., Solanaceæ. Native of India and Africa. The plant is a biennial, about 2 or 3 feet high. It is clammy and has a disagreeable odour. Its flowers are yellow with dark purple veins. The leaves are sinnated and hairy.

Analysis.—Two alkaloids are present—Hyoscyamina, $C_{17}H_{23}NO_3$: Hyoscina, $C_{17}H_{21}NO_4H_2O$. Children bear hyoscyamus well, but the aged do not.

Antidotes.—Stomach pump or emetic. Stimulants. Coffee enemata. Hypodermics of pilocarpin. Artificial respiration.

IGNATIA BEANS.—Strychnos Ignatii. Nat. Ord., Loganiaceæ. Native of Philippine Islands. A large climber with ovoid seeds bluntly angular or flattish and covered with silvery hairs. It has conspicuously three-nerved elliptic leaves. The fruit is globose or ellipsoid and about 4 inches long.

Toxic principles.—Strychnina and Brucia.

Antidotes, as for strychnine.

INDIAN HEMP.—See *Cannabis sativa.*

ISOTOMIA LINEARIS.—Nat. Ord., Lobeliaceæ. Native of Brazil, now found everywhere in tropics. Is a small herb with star-shaped white flowers. Pungent and hot to the taste.

Active principle probably a glucoside, lobelin. It is said to have an inflammatory renal action.

The *treatment* should be conducted on general principles, such as emetics, stimulants (if required), &c.

JABORANDI. — Pilocarpus pennatifolius. Nat. Ord., Rutaceæ. Native of Eastern Brazil, not found elsewhere. An evergreen shrub, 4 or 5 feet high with compound imparipinnate leaves; leaflets oblong, coriaceous, 3 inches long by 2 inches wide, showing translucent oil glands by transmitted light, and somewhat resembling the leaf of the cherry laurel.

The active principles is found in the leaves, and is an alkaloid called "Pilocaripina," $C_{11}H_{16}N_2O_2$, one of the few liquid alkaloids.

Action and Uses.—Jaborandi is an active diaphoretic, and is the most powerful sialogogue known.

Antidote.—Belladonna by the mouth, or atropine gr. $\frac{1}{60}$. hypodermically repeated in twenty minutes if necessary.

JAVA BEANS.—Phaseolus lunatus. Nat. Ord., Leguminosæ. Origin not known. Now ubiquitous in tropics. Is the original and wild form of the haricot-bean. It is an annual climber with white flowers, and with brown or purple beans. (The cultivated species known as the haricot-bean is a white bean, and is innocuous.) The beans have been used for green-soiling, and eaten by mistake. Fatal results have occurred. The import of these beans into France is prohibited.

The *toxic agent* is hydrocyanic acid, produced from the glucoside phaseolunatin.

Antidotes. — Emetics. Atropine gr. $\frac{1}{60}$ hypodermically, and repeated in twenty minutes if necessary.

KECHUBONG.—See *Datura fastuosa.*

MANCHINEEL.—Hippomane mancinella. Nat. Ord., Euphorbiaceæ. Allied to Excæcaria. A West Indian tree possessing an acrid milky latex. The fruit is said to be like an apple. Both latex and fruit are poisonous, and have been used criminally. The tree does not occur in the East. The toxic agent is unknown, but is probably a glucoside.

MANIHOT UTILISSIMA.—See *Cassava.*

MUSHROOMS.—Agaricus. Class, Fungi. Nat. Ord., Basidiomycetes. Species ubiquitous, some of them being toxic. The tropical agarici have not been worked out. The poisonous principle is an alkaloid amanitina or muscarina, $C_5H_{15}NO_3$.

Antidote. Atropine gr. $\frac{1}{60}$ hypodermically, and repeated in twenty minutes if necessary.

NERIUM ODORUM.—See *Oleander.*

NUX VOMICA. See *Strychnos.*

OLEANDER. Nerium odorum—the sweet-scented oleander. Nat. Ord., Apocynaceæ. Hindu name "Kaner." Is a native of India, used in external medication for leprosy and skin diseases. Roots highly poisonous and used in India both for suicidal purposes and to procure abortion.

Toxic agent said to be an alkaloid "Neriodorin." Causes profound depression of the central nervous system.

Antidotes.—Emetics. Stimulants. Strychnine hypodermically.

PACHYRRHIZUS TUBEROSUS.—See *Yam bean.*

PHASEOLUS LUNATUS.—See *Java bean.*

PHYLLANTHUS URINARIA.—Nat. Ord., Euphorbiaceæ. A small weed 6 or 8 inches high, with small oblong leaves. Flowers and fruit extremely small.

Is ubiquitous in the tropics and common on waste grounds. Used in native medicine for diuretic action In large quantities causes renal congestion. Eaten by children and lunatics accidentally. Toxic principle not known.

PHYSOSTIGMA VENENOSUM.—See *Calabar bean.*

PILOCARPUS PENNATIFOLIUS.—See *Jaborandi.*

PLUMBAGO ROSEA.—Nat. Ord., Plumbaginaceæ. Native name, "Cheraka merak." Origin not known. Ubiquitous distribution in tropics. Is an ornamental herb with red flowers.

Toxic Agent.—A glucoside called Plumbagin, which is found in the leaves and whole stem.

Action and Uses.—It is used as a vesicant for rheumatism and leprosy, also to procure abortion, being either taken internally (as a decoction) or more commonly introduced into the vagina, and sometimes into the uterus, naturally frequently producing pelvic peritonitis.

RENGAS.—"Gluta renghas," "Gluta coarctata." Nat. Ord., Anacardiaceæ. Native of Malayan Peninsula and Archipelago. Not found elsewhere in the tropics. The plants of this Cashew-nut family generally contain an acrid-resinous juice. A slight amount of the same principle is said to be contained in the mango.

Rengas are trees with a thin greenish slimy latex, soon becoming black and viscid, which produces considerable erythema and œdema if applied externally, and a fatal enteritis if taken internally. It is of no economic use, but is occasionally used criminally. The toxic agent is unknown.

RICINUS COMMUNIS.—See *Castor-oil.*

ROTAN.—Calamus. Nat. Ord., Palmaceæ. A climber found in most of the tropics, except S. America. Scrapings are administered criminally, usually mixed with powdered glass. They can be detected under the microscope by the oblong or square siliceous cells, and small pieces containing stomata.

SASSY-BARK.—Erythrophlœum guineense. Nat. Ord., Leguminosæ. A native of West Africa. Not found elsewhere. The bark is reddish-brown, hard, brittle, and astringent. Medicinally used as an astringent, an emetic, a narcotic, and as a local anæsthetic. Is used as an ordeal poison in W. Africa. The toxic agent is a glucoside, "Erythrophlœin," about which little is known.

STRYCHNOS.—Nux vomica. Nat. Ord., Loganiaceæ. A native of India, Ceylon, Coromandel, &c. Is a tree of middle size. Its fruit is the size of a large apple, smooth externally, and filled with a soft white pulp, in which the seeds are embedded. The seeds are nearly circular and flat, about 1 inch in diameter and ¼-inch thick. They are umbilicated on one side, ash-grey colour externally, thickly covered with short satiny hairs, internally translucent, tough and horny, and taste intensely bitter, without odour.

Toxic principles are two alkaloids—Strychnina, $C_{21}H_{22}N_2O_2$, and brucia, $C_{23}H_{26}N_2O_4 . 4H_2O$.

Action.—The medulla is the part affected. Violent tetanic spasms occur, without affecting the sensorium.

Antidotes.—Emetics, pot. brom., chloral, amyl nitrite or chloroform inhalations. Curare hypodermically.

STRYCHNOS IGNATII.—See *Ignatia beans.*

TANGHINIA VENENIFERA.—Nat. Ord., Apocynaceæ. Native of Madagascar. Limited distribution. It is a tree somewhat re-

sembling Cerbera, but with narrower leaves. Its bean is used as an ordeal bean.

The *toxic agent* is a colourless crystalline neutral principle called Tanghinin, allied to Strophanthin, and having the same physiological effect.

Antidote.—Emetics, stimulants, hypodermics of strychnine.

THEVETIA NERIIFOLIA.—See *Yellow oleander.*

TUBA.—Derris elliptica. Nat. Ord., Leguminosæ. Native of the Malayan Peninsula and Archipelago and the East Indies. Is a climbing plant, cultivated as an insecticide and piscicide. Bark in long strips of a dark brown colour. The roots are long, smooth, and dark brown, and of various diameters, having a nauseous taste. Decoctions have been occasionally used criminally and for suicide, but as large quantities are required, it is seldom used. It is also used as an abortient, the root being inserted and left in the vagina, causing a metritis.

The *toxic agent* is a glucoside called Pachyrrhizin (or Derrid, or Tubain), and is met with also in the Yam bean (which see).

YAM BEAN.—Pachyrrhizus tuberosus. Nat. Ord., Leguminosæ. Native of the West Indies. Cultivated all over tropics. Native name, "Bengkawang." The tuber is round, like a black radish, and is used as an edible vegetable, for which the plant is cultivated. The pods are green beans, much like a French bean in appearance, containing five or six small black seeds. When ripe these seeds are poisonous owing to a glucoside, Pachyrrhizin (see *Tuba*). Deaths have been caused by the accidental use of these seeds.

YELLOW OLEANDER.—Thevetia neriifolia. Nat. Ord., Apocynaceæ. Native of America and West Indies. Naturalised and cultivated in India and Malaya. Native names, "Zard kunel" (Hindu) and "Pach-ch-ai-alari" (Tamil). The latex of the bush is highly poisonous, as also the bark and kernels. Medicinal use as a febrifuge. Toxic doses produce vomiting and purging.

Toxic Agent.—An alkaloid, Thevetina. Composition doubtful.

SECTION II.

Injection Poisons.

ACOKANTHERA.—Also used as an internal poison (see previous section).

ANTIARIS TOXICARIA.—Upas tree. Nat. Ord., Urticaceæ. Malay name, "Ipoh." Native of Malayan Archipelago. The inspissated latex is used by wild tribes for poisoning darts, in combination with Strychnos.

Toxic Agent.—A brown gum-resin, "Antiarin," harmless when taken by mouth, but fatal when injected, producing violent intestinal peristalsis. Cases of accidental death have been recorded.

Antidotes are not of much use, as it is fatal in three to five minutes. Pot. brom., chloral, or morphia injections.

CRAB'S EYES.—Abrus precatorius. Nat. Ord., Leguminosæ. Synonyms—Jequirity seed; false liquorice. A small climbing vetch. Red seeds with black spots. Roots used medicinally in place of liquorice seeds in granular conjunctivitis. Seeds contain a glucoside, "Abrin." Toxic only when injected. Used in India for cattle-poisoning.

CURARE.—Malonetia nitida. Nat. Ord., Apocynaceæ. Synonyms—Ourari, wourara, wourali, and urari. Is a S. American arrow poison (being a decoction and extract of the plant, together with various species of Strychnos, &c.). Is imported from the Amazon region as a black paste, with no smell, and not poisonous if taken internally. If injected, it paralyses the peripheral ends of the motor nerves of the voluntary muscles, and has, therefore, been used in tetanus.

The *poisonous principle* is an alkaloid, Curarina, $C_{18}H_{35}N$.

Antidote.—Strychnine.

STROPHANTHUS HISPIDUS.—Nat. Ord., Apocynaceæ. Native of Africa. Native name, "Kombe." The fruit is a follicle about 12 inches in length, and the seeds are remarkable for their appendages, which give them an arrow-like appearance. The seeds contain a colourless, crystalline, neutral principle called Strophanthin. It is a cardiac stimulant, and used by natives as an arrow poison.

STRYCHNOS.—Is also used as an internal poison (see previous section).

STRYCHNOS TIEUTE.—Nat. Ord., Loganiaceæ. Malay name, "Ipoh akar." Native of Malay Peninsula. Is a climber with deep green leaves, and curious round ball-like fruit of a greyish-green colour. Native use, a decoction of the bark mixed with Antiarin, and used as an arrow poison. Prepared as a black paste with intensely bitter taste.

Toxic Agent.—Brucia.

Antidotes.—Pot. brom. and chloral.

SECTION III.

Drug Poisons.

CAMPHOR.—Cinnamomum camphora. Nat. Ord., Laurineæ. A large tree, 30 ft. high, with beautiful evergreen leaves. Is a native of China, Japan, &c.

From the wood is obtained a stearoptene—Camphora—and purified by sublimation.

Is a white, translucent, tough, and crystalline body, with powerful odour and pungent taste; floats on water; volatilises slowly at ordinary temperatures; slightly soluble in water; readily soluble in alcohol and ether. Dose 1 to 10 grains.

Toxic amount causes giddiness, stupor, delirium, convulsions.

Antidotes.—Stomach tube, stimulants, and warmth to extremities.

COCA.—Erythroxylon coca. Nat. Ord., Lineæ. A small shrub, 4 to 6 ft. high, with numerous spreading branches. Native of Peru and Bolivia. Leaves are shortly stalked, oval or lanceolate; 1 or 2 inches in length, entire, quite smooth; mid-rib prominent; green above, paler beneath. Odour faintly tea-like. taste bitter and aromatic.

From the leaves is obtained an alkaloid Cocaina, $C_{17}H_{21}NO_4$. Dose gr. $\frac{1}{5}$ to $\frac{1}{2}$.

Symptoms of toxic dose:—Pallor, dry skin, giddiness, quickened respiration, tremors, and convulsions.

Antidotes.—Stomach tube, stimulants, morphine hypodermically, amyl nitrite inhalations, artificial respiration.

COLOCYNTH.—Citrullus colocynthis. Nat. Ord., Cucurbitaceæ. The plant is succulent, hairy, and procumbent, like the cucumber family generally. It has small yellow flowers, and globular smooth fruit about the size of an orange. Native of S.-E. Europe, Asia, and Africa.

Medicinal use is made of the dried, decorticated fruit, freed from seeds, on account of its bitter principle—a glucoside Colocynthin—to which it owes its purgative properties as a hydragogue cathartic.

Toxic doses produce gastritis, enteritis, and colitis.

Antidotes.—Wash out stomach with olive oil, 4 ozs. to 1 pint water. Demulcent drinks, as white of egg in milk, morphine hypodermically. Stimulants as required.

OPIUM.—Papaver somniferum. Nat. Ord., Papaveraceæ. Native of S.-E. Europe and Asia. Differs from other poppies in being quite glabrous.

Opium is the inspissated juice, obtained by incision of the unripe capsules. Has a peculiar odour and nauseous, bitter taste. It is one of the most complex substances used in medicine, containing two acids, two neutral principles, and nineteen alkaloids, the chief of which, however (10 per cent.), is Morphina, $C_{17}H_{19}NO_3H_2O$. Is a narcotic poison, in *toxic doses* causing giddiness, depressed circulation and respiration, contraction of pupils, coma, and death.

Antidotes.—Stomach pump, emetics, smelling salts, brandy and coffee, artificial movements and respiration. Pot. permang. gr. viij. in water taken internally, and repeated if necessary.

SECTION IV.

Chemical Analysis for Alkaloids.

The substance being an organic solid or mixture:—

1. Acidify with a little acetic acid.
2. Digest with water for one hour.
3. Filter.

4. Evaporate filtrate to a syrup.
5. Mix this syrup with three times its volume of alcohol.
6. Distil again to consistency of a thin syrup.
7. Cool and digest with water. (Fats and resins separate.)
8. Filter through wetted paper.
9. Add lead acetate as long as ppt. falls (decolourises).
10. Pass H_2S (throws down the lead).
11. Filter and evaporate to consistency of an extract.
12. Treat with 50 c.c. of alcohol (mucus and dextrins precipitated).
13. Filter.
14. Evaporate alcohol.
15. Take up residue with 50 c.c. water.
16. Filter.

Small portions of filtrate may now be concentrated and tested by general test.

To 1 drop add 1 drop Mayer's reagent. *Ppt. = alkaloid*, and proceed.

If no precipitate with Mayer, add to another drop 1 drop of H_2SO_4 conc., 1 drop $K_3Fe_2Cy_6$ and 1 drop $FeCl_3$. *Blue ppt. = Ptomaine.*

Note.—Morphine and aconitine will both give this blue precipitate, but they will have been excluded by absence of precipitate with Mayer's solution.

17. Shake filtrate with *petroleum ether*. Separated :—Digitalin, antipyrin, phenacetin, salicylic acid, picric acid.

After removing petroleum ether :—

18. Make strongly alkaline with NaOH.

19 Shake with *Benzine*. Separated :—Strychnine. brucine, emetine, quinine, atropine, hyoscyamine, aconitine, codeine. &c.

After removing the benzine

20. Shake with hot *amyl alcohol*. Separated :—Morphine, apomorphine.

Having extracted the alcohol by one of the above solvents, the following individual tests will show the nature of the alkaloid :—

Dissolve a grain or so of the alkaloid. glucoside. &c., in a few drops of water, or dilute HCl.

				Confirm by
To a small quantity on a porcelain plate add one drop HNO_3 conc.	Purple-red	=	*Apomorphine.*	$FeCl_3$ = deep red.
	Blood-red	=	*Brucine.*	
	Orange-red	=	*Codeine.*	
	,, ,,	=	*Morphine.*	Fröhde's reagent gives intense violet at once.
	Dirty red	=	*Veratrine.*	See next section.

				Confirm by
To another portion add H_2SO_4 conc.	Red or brown (deep red if warmed)	=	*Veratrine.*	HCl conc. = red-purple.
	Bluish	=	*Codeine.*	$H_2SO_4 + FeCl_3$ = blue-black, becoming scarlet and orange on adding HNO_3 dil.
	Blood-red	=	*Salicin* (glucoside).	
If not found, heat in dry tube.	Red vapours	=	*Quinine.* *Quinidine.* *Cinchonine.* *Cinchonidine.*	

If still no result, test specially as follows:—

I. *If an alkaloid*:—

Aconitine.—One drop dilute solution on tongue = numbness and tingling.

Atropine.—Alcoholic sol. + alcoholic sol. $HgCl_2$ and warmed = red precipitate.

Caffein.—Add $KClO_3 + HCl$. On adding NH_3 = purple-red colour.

Cocaine.—(1) Add K_2MnO_4 = purple precipitate. (2) Boil c̄ KOH. Neutralise c̄ HCl = crystalline precipitate of benzoic acid. (3) In presence of other alkaloids may be detected by giving yellow precipitate c̄ K_2CrO_4 in presence of free HCl.

Physostigmine.—Warmed c̄ KOH gives red colour, becoming bluish on evaporation to dryness.

Pilocarpine.—H_2SO_4 conc. gives yellowish solution, becoming emerald-green on addition of $K_2Cr_2O_7$.

Strychnine.—Place one drop or small fragment of strychnine on a porcelain plate, and near it a small fragment of $K_2Cr_2O_7$. Add to each a drop of H_2SO_4 conc. Then draw the latter over the strychnine spot, when a beautiful evanescent purple is produced.

II. *If not an alkaloid*:—

Acetanilide.—Heat c̄ KOH and $CHCl_3$ = unpleasant odour of phenyl carbamide, C_6H_5NC.

Phenacetin.—Boil c̄ HCl. Dilute, cool, filter, add $K_2Cr_2O_7$ = deep red.

Antipyrin.—To aqueous solution add $NaNO_2 + H_2SO_4$ dil. = deep green.

* *Picrotoxin* (glucoside) occurs in Cocculus indicus and Anamirta paniculata.

Should be crystallised out, and crystals compared with type.

* *Digitalin.*—Add HCl = faint yellow, rapidly green.

Add H_2SO_4, and expose to vapour of bromine = violet colour.

* *Colocynthin.*—Soluble in water and alcohol, not in ether.

* *Strophanthin.*—Add $FeCl_3 + H_2SO_4$ conc. = red-brown precipitate, turns green in one or two hours.

Add H_2SO_4 = dark green, turning to red brown.
Add phospho-molybdic acid = bright blue-green colour.

Note.—These glucosides when heated with dilute acid yield glucose, which will reduce Fehling's solution to the yellow oxide.

FORMULÆ.

Fehling's Solution.—

$CuSO_4$, . .	8·66 grammes.	}	Mix when required, and make up to 250 c.c. with water.
Aq. dest., . .	62 c.c.	}	
K. Na. Tart., .	43 grammes.	}	
Aq. dest., .	120 c.c.	}	

Mayer's Reagent.—Potassio-mercuric iodide ($HgI_2 2KI$) made up in decinormal strength.

Fröhde's Reagent.—Five milligrammes of sodium molybdate are dissolved in each c.c. of strong H_2SO_4, according to the amount of stock solution required.

Note.—In all the above manipulations, one drop of the reagent is usually sufficient.

APPENDIX III.

HOW TO COLLECT BLOOD-SUCKING FLIES, TICKS, &c.

British Museum Pamphlet, by E. E. Austen, F.Z.S., Dipterologist to Nat. Hist. Museum.

Directions for the Collection of Specimens.

List of Articles Required.

An Entomologist's Collecting-net.—This can be obtained from any dealer in natural-history apparatus. Any net used for collecting butterflies will do for Diptera; but, on the whole, perhaps an ordinary umbrella-net will be found the most serviceable. One or two spare net-bags should be taken in case the one in use gets torn.

Two dozen glass-bottomed *cardboard pill-boxes* (assorted sizes, up to 2¼ inches in diameter, packed in nests one inside another).*

One or two *cyanide killing-bottles*, not too large to be carried in the pocket when required; or a larger-sized cyanide killing-jar, or materials for making same, as follows;—¼ lb. of cyanide of potassium,† 1 lb. of plaster of Paris, a glass jar with wide mouth and closely-fitting lid.‡

* These boxes can be obtained from Messrs. Watkins & Doncaster, 36 Strand, London, W.C.; but care should be taken to see that the *bottoms*—and not the tops, as is often the case—are made of glass. Since the boxes are constructed of cardboard, they are liable in tropical countries to go to pieces in the rains; and to prevent this they should be covered with jaconet in the following manner, the important point to remember being that the jaconet must be cut in strips *on the cross*:—Obtain, say, a square yard of the material, and fold it into a triangle by bringing two opposite corners together. Consider how wide the strips must be, according to the varying depths of the boxes to be covered, and rule them off in pencil by drawing lines *parallel to the base* of the triangle. Cut up the strips, or, if possible, get them cut by a bookbinder's machine. Paint the box over with liquid glue, and wrap the jaconet round it: it is particularly important that the edges of the glass and lid should be well protected; and it will be found that by gently pulling the jaconet it will wrap itself round these without difficulty. When quite dry, say the following day, the box should be given a coat of Aspinall's enamel, or of paint, a second (and if necessary a third) coat of paint being added after the first is dry. Any paint or glue on the glass can be removed with a penknife; if the jaconet protrudes too far over the glass, cut it round with a penknife and remove it. (Glass-bottomed boxes already protected in this manner can be obtained from Miss E. M. Bowdler Sharpe, Entomological Agency, 4 Barrowgate Road, Chiswick, London, W.)

A simpler method of protecting the boxes is to coat them (especially the joints) with shellac dissolved in absolute alcohol.

Glass-bottomed boxes of this kind constructed of tin are sometimes sold; but these are not to be recommended, since when in use in warm climates they are apt to become very hot, with the result that flies contained in them are killed and become dry prematurely.

† If it is intended to take cyanide to a damp tropical climate, it should be conveyed in the form of *lumps*, in a bottle with a tightly-fitting glass stopper. Cyanide of potassium is also sold in rods, and, in this form, might be conveniently carried in short lengths in hermetically-sealed tubes of thin glass, of diameter and length just sufficient to take the section of cyanide rod.

‡ Cyanide killing-bottles can be procured ready for use from Hinton & Co., Bedford Street, London, W.C., or any other chemist will prepare one to order; but when Diptera are collected in the manner advised below, it is preferable to use a large-sized killing-jar, which should be made as follows:—Take any fairly large glass jar (such as a pickle-bottle) with

Entomological forceps, two pairs (Fig. 96), with curved ends for holding pins (from G. Buck, 242 Tottenham Court Road, London, W.).

Fine-pointed Forceps (one or two pairs).—These are useful for arranging the legs and wings of specimens when pinned; they can be obtained with the above.

Needles (two or three) *mounted in handles*—also for arranging legs and wings.

Entomological pins (D. F. Taylor & Co., New Hall Works, Birmingham), Nos. 5 (1s. 6d. per ounce), 7 (2s. 6d. per ounce), and 20 (7s. 6d. per ounce). The No. 20 pin should be used for all but the very largest Diptera, such as Horse-flies (Tabanidæ); as it is exceedingly fine, an ounce will go a very long way.

Fig. 96. Entomological forceps.

Common pins—a thousand or two, in paper packets.

Gun-wad punches, Nos. 4, 12, and 20 bores (from any gun-maker), for punching discs of card.

Cards (3-sheet Bristol board), from which to punch discs; a supply of the latter should be prepared ready for use.

A platyscopic lens (Messrs. Baker, 244 High Holborn, London, W.C.; or John Browning, 63 Strand, London, W.C.; price about 15s.). The magnifying-power should not be too high—from 10 to 15 diameters is about the best.

Cork-carpet or pith.—Two or three sheets about 6 inches square,

a wide mouth and closely-fitting lid (a lever-lid such as those often fitted to pickle-bottles would answer admirably), and cover the bottom with a layer of dry plaster of Paris to the depth of ½ inch; pour in above this a layer equal in depth consisting of *powdered* cyanide of potassium, mixed with rather more than its bulk of dry plaster of Paris; cover this mixture with a layer of dry plaster of Paris to the depth of ¼ inch or so; and pour in above the whole a layer ½ inch in depth, consisting of plaster of Paris mixed with water to the consistency of cream. As soon as the top layer of plaster is dry the jar is ready for use: the plaster, however, should be covered with several thicknesses of blotting-paper, to avoid risk of injury to specimens in case the surface should at any time become wet. To obviate the danger of cracking the jar owing to the heat evolved when plaster of Paris is mixed with water, it may be advisable to stand the jar in warm water before adding the final layer. The exact amount of cyanide of potassium to be used is of no great consequence; but in the case of a properly-prepared jar the odour should be readily perceptible on removing the lid: if it is not, the reason may be that the mixture is too dry, when a little water poured on to the top layer will probably set matters right. After some months' use the cyanide loses its efficacy (to obviate this so far as possible the jar should never be allowed to remain open), and the mixture must then be renewed.

A rough-and-ready method of making a killing-jar or bottle is to cover the bottom with a layer of powdered cyanide, and to place above this a number of layers of blotting-paper. The layers of blotting-paper immediately in contact with the cyanide must be slightly damped; but only sufficient water should be used to cause the cyanide to give off its odour. The top layer of blotting-paper must on no account be wet, and the less water used the better.

on which to perform the operations of pinning, &c. (Cork-carpet can be obtained at Harrod's Stores, Brompton Road, London, S.W.)

Two or three cork-lined entomological store-boxes.—These can be obtained from Messrs. Watkins & Doncaster, or any other dealer in natural-history apparatus. For a collecting trip or expedition of some duration the boxes should not be smaller than about 18 inches by 12, and they must be sufficiently deep to prevent the heads of the pins from coming into contact when both sides of the box are filled. Should the collector run out of store-boxes, *cigar-boxes*, in the bottom of which is fixed a layer of cork-carpet or pith, make efficient substitutes; but if pith is used, it should not be less than ½ inch thick.

Method of Collecting.

If possible, Diptera should always be brought home alive in the glass-bottomed pill-boxes (to which they are to be transferred on being captured in the net), and should then be killed in the cyanide-bottle or jar immediately before being pinned. As soon as a fly is taken in the net by a dexterous sweep, a sharp turn of the wrist must be given (following a smart downward or lateral stroke in order to bring the fly to the end of the net), in such a way that the end of the net containing the insect falls over the rim and so makes a closed bag from which it cannot escape. The end of the net can then be gathered up in the hand, and the fly forced into a still smaller space, in which it will not be difficult to get it into a pill-box, and then to slip on the lid. If the specimens are small, it is possible with care to get several into one pill-box. Flies may also be transferred direct from the net to the killing-bottle, and so brought home dead; but this method is not to be recommended, since prolonged exposure to the effects of cyanide of potassium is apt to injure the specimens, the ultimate condition of which, when so treated, is rarely as satisfactory as if they had been brought home alive in pill-boxes. If, however, it is necessary for any reason to dispense with pill-boxes, and to use the killing-bottle in the open, a little crumpled tissue-paper should be placed inside it; this affords a lodgment for the specimens, and so lessens the risk of their being injured by rolling about. It is always advisable when out collecting to carry a killing-bottle for use in case of need, in the event of the supply of pill-boxes running short. Diptera on windows may be captured in pill-boxes; if the edge of the box is slightly raised from the glass on one side, and a little tobacco smoke blown into it so as temporarily to stupefy the fly, it will be found easy to slip on the lid without allowing the insect to escape.

Killing.

Diptera brought back alive after a day's collecting should be killed by being placed for a few minutes in the closed cyanide-bottle

or jar. If the cyanide-jar is sufficiently large, the pill-boxes themselves may be placed therein, first opening them a fraction of an inch on one side to allow the cyanide to take effect. In the case of large sluggish Diptera, such as *Tabanus*, the pill-box may be opened without fear of the insect effecting its escape; after which a smart tap on the bottom of the box will cause the fly to drop into the jar. Diptera should never be allowed to remain exposed to the effects of the cyanide longer than is necessary to ensure their being quite dead. If the poison is of reasonable strength, four or five minutes should be sufficient to kill even the largest and strongest flies; on the other hand, it is necessary to make sure that the insects are really dead, since, if the exposure to cyanide has been too short, flies will often appear to be dead when in reality they are only stupefied. As soon as the insects are really dead, they should be removed from the killing-jar; those in pill-boxes may be allowed to remain therein until one is ready to pin them, while the loose specimens should be turned out on to a sheet of cork-carpet or pith.

Methods of preserving Diptera other than by Pinning.

Diptera should always be pinned, and this should be done as soon as they are dead. If preserved in any other way, they will never make such satisfactory specimens.

When, however, it is impossible to pin them, Diptera may be preserved in fine sawdust, on which a few drops of dilute carbolic acid should be sprinkled to prevent mould. Each specimen, before being placed in the sawdust, should be loosely screwed up in fine tissue-paper (cigarette-paper would do), on which the necessary data (see below under "Pinning") should be written in pencil. To contain the specimens a small tin box (such as those in which tobacco is sold) should be used; and to prevent injury from shaking, the box should be packed *quite full* of sawdust and specimens.

Diptera may also be put away for transport in three-cornered envelopes of soft paper (newspaper), after the manner in which butterflies are packed by collectors; but this method does not suit thick-bodied flies, such as *Tabanidæ*, as these are often crushed by it, and if once flattened cannot be restored to their natural shape. Envelopes containing Diptera or other insects may be conveniently packed in tin tobacco- or biscuit-boxes for transmission by parcel post from abroad. A few drops of weak carbolic acid should be sprinkled over each layer of envelopes, and the box should be packed quite full.

It is never advisable to put Diptera, intended for transmission by post, into small boxes containing wool, as when flies are once in contact with wool, and have become dry, it is very difficult to remove them without pulling off legs, bristles, &c.

Pinning.

Take a card disc (Fig. 97), and write on it all the data connected with the specimen to be pinned, as follows :—(1) name of *locality*, including altitude if necessary; (2) *date*—day, month, year—thus, 6. 3. 04; (3) *collector's name*; (4) any *brief remarks of interest* (which may if necessary be written on the other side)—*e.g.*, "Very common"; "Only specimen seen"; "On porter's back"; "At watering-place in stream"; "Running on banana leaves." Longer notes on habits, &c., should be entered in a note-book, with a number corresponding to one written on the disc. Specimens taken *in coitû*, for which a good look-out should always be kept when collecting, should have the fact noted on the disc of each, thus :—"A—*in coitû* with B"; "B—in *coitû* with A."

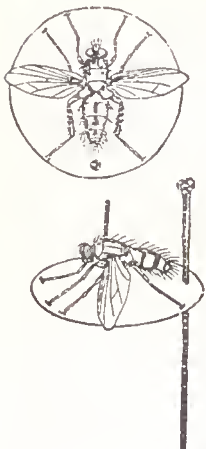


Fig. 97.—Use of discs.

Place the disc on which the above particulars have been written, plain side uppermost, on a sheet of cork-carpet or pith, and, picking up with the forceps an entomological pin (a No. 20 if the specimen is not larger than a Blue-bottle, otherwise a No. 7 or No. 5), thrust it through the centre of the thorax of the specimen, until about $\frac{1}{3}$ inch protrudes beneath. Next grasp the pin with the forceps near the tip, and thrust it through the disc, drawing it well down. Lastly, thrust an ordinary pin through the disc near the margin for the purpose of carrying both disc and specimen, and draw the disc a good half-way up the carrying-pin.

The last thing to be done is to arrange the legs and wings as far as possible. The wings must be made to project at an angle from the body, and not allowed to lie closed over it; if they can be got to remain at right angles to the body, so much the better. In the case of any specimen that is not too small and fragile, the wings can be best arranged by means of the fine-pointed forceps, by making a gentle simultaneous pressure with the tips of the forceps at the base of each wing, repeating it until the wings assume the desired position. The legs also must be disposed symmetrically (and as far as possible in a natural position) on the card disc, so that all parts of them can be readily seen, and must not be allowed to remain crumpled up beneath the body, since important characters are often found upon them. In the case of a fairly large specimen it will generally be found possible to cause the legs to remain in the desired position by hooking the claws on the edge of the disc, gently drawing the legs out one after another by aid of a needle or one leg of the fine-pointed forceps. The manipulations in connection with the legs and wings

must be performed as gently as possible, and care must be taken that bristles, hairs, or scales are not rubbed off in the process. As soon as these operations are completed, the specimen should be transferred to a store-box, or to one lined with cork-carpet or pith, as described above. Since, however, the tissues contract in dying, the legs and wings are very apt to get pulled out of place, and, to correct these changes, the specimens should be examined once or twice during the next day or two after being pinned.

Very minute or fragile specimens (such as *Ceratopogon* or *Simulium*) are best *pinned from the side*, and the legs should then be straightened out by means of a No. 20 pin held in the entomological forceps.

Diptera Collected in the Tropics—Attacks by Ants—Mould—Transmission to England.

In the tropics boxes of pinned insects are very liable to the attacks of minute *ants*, which, if they once gain access to a box unobserved, will soon play havoc with its contents. In Brazil it has been found that ants can be prevented from entering insect-boxes by smearing the outside round the line where the box opens, or any other possible place of entrance, with *andiroba* oil, repeating the process as often as necessary; similar methods might be employed elsewhere in case of need.

Mould, however, is an even greater enemy to collections of Diptera. Pinned specimens of Diptera, like those of other insects, rapidly develop mould during the rainy season in tropical countries; and since mouldy specimens are practically worthless for purposes of scientific determination, *Diptera should always be sent home as soon as possible after being collected.* The risk of mould may, however, be diminished by pinning in a corner of the box a small piece of sponge saturated with the strongest carbolic acid, which should be constantly renewed. The greatest care must be taken to prevent specimens getting loose and rolling about in transit, since in this way a single loose disc might easily do irreparable injury to many other specimens in the box. To prevent this the pins supporting the discs should be inserted as tightly as possible into the cork-carpet or pith, and they should all be driven into the same level, after which a sheet of soft paper (newspaper does very well) can be *fixed* into the box, resting on the heads of the supporting pins, in order to minimise the damage should a disc happen to get loose. The box containing the specimens should be well wrapped in cotton-wool or similar material, to secure it from shocks on the journey, and firmly *packed in an outer case* for transmission (by parcel-post if possible and the package is not too large) to England.

Number of Specimens of each Species Required.

At least half a dozen specimens of *each sex* of a species should, if possible, always be obtained, and a good look-out should be kept for specimens showing any abnormality in structure, colouration, or

size. When the collector remains long enough in one spot, he should always endeavour to obtain specimens of a species on different dates, in such a way as to throw as much light as possible on the duration of its seasonal occurrence. Similarly the attempt should be made to illustrate the geographical range or local distribution of a species, by collecting specimens in as many different localities as possible. Specimens of species taken in new localities, though common elsewhere, will always be valuable.

In addition to Pinned Specimens, others should be Preserved in Alcohol or Formalin.

Pinned specimens should always, if possible, be accompanied by others preserved in alcohol or formalin. If the latter preservative be used, a 2 per cent. solution will probably be found of sufficient strength. In the case of alcohol, the spirit used should not at first be stronger than 20° below proof; after the specimens have been immersed in this for two or three weeks, the strength of the spirit should be raised to 20° above proof, in which the insects may be kept and forwarded to England. The specimens should be preserved in small glass tubes, with a plug of cotton-wool placed inside on the top of the specimens to prevent them from being injured by washing about in transit. A slip of paper, on which all necessary data should be written clearly *in pencil*, should be placed *inside* each tube, and the pinned specimens of the same species should be labelled with a reference to those in spirit, so that the latter may be identified.

Larvæ.

Specimens of larvæ will always be valuable, whenever it is possible to breed out some of them so as to determine the fly to which they belong. Unless, however, the perfect insect is known, it is rarely possible to do more than to determine the *family* or *genus* to which a larva belongs. Larvæ for preservation should be killed by immersion for a *moment or two* in boiling water, and should then be placed in weak spirit (two-thirds spirit and one-third water); after being allowed to remain in this for two or three weeks until thoroughly hardened, they can be transferred to stronger spirit. The larvæ of each species must of course be kept separate, and should be put up in a small corked glass-tube full of spirit, the necessary particulars, with, if possible, a reference to pinned specimens of the perfect insect, being written in pencil on a scrap of paper and placed *inside*.

Notes on Habits, &c.

Comparatively little is yet known as to the bionomics of Blood-sucking Diptera in general, so that notes on the habits, distribution, seasonal occurrence, relative frequency, &c., of particular species will always be of great value and interest. Clinical observations as to the effect of the bites of the various species on man and domestic animals are also required.

APPENDIX IV.

POSOLOGICAL TABLE.

POSOLOGICAL TABLE OF THE COMMONER DRUGS.

Note.—Drugs marked * are non-official.

	IMPERIAL.				METRIC.			
Acetanilidum (Antifebrine),	gr.	1 to gr.		3	0·065	gm. to	0·194	gm
Acidum Arseniosum,	,,	$\frac{1}{60}$	,,	$\frac{1}{15}$	0·00108	,,	0·0043	,,
,, Benzoicum,	,,	5	,,	15	0·324	,,	0·972	,,
,, Boricum,	,,	5	,,	15	0·324	,,	0·972	,,
,, Camphoricum,	,,	10	,,	20	0·648	,,	1·296	,,
,, Carbolicum.	,,	1	,,	3	0·065	,,	0·194	,,
,, ,, Liquefactum,	min.	1 to min.		3	0·059	c.c. to	0·178	c.c
,, Citricum,	gr.	5 to gr.		20	0·324	gm. to	1·296	gm
,, Gallicum,	,,	5	,,	15	0·324	,,	0·972	,,
,, Hydrobrom. Dil.,	min.	15 to min		60	0·888	c.c. to	3·55	c.c
,, Hydrochlor. Dil.,	,,	5	,,	20	0·296	,,	1·184	,,
,, Hydrocyanic. Dil..	,,	2	,,	6	0·118	,,	0·355	,,
,, Lacticum,	,,	5	,,	20	0·296	,,	1·184	,,
,, Nitricum Dil.,	,,	5	,,	20	0·296	,,	1·184	,,
,, Nitrohydrochlor. Dil..	,,	5	,,	20	0·296	,,	1·184	,,
,, Phosphoric. Dil.,	,,	5	,,	20	0·296	,,	1·184	,,
,, Salicylicum,	gr.	5 to gr.		20	0·324	gm. to	1·296	gm
,, Sulphuricum Aromaticum.	min.	5 to min.		20	0·296	c.c. to	1·184	c.c
,, Sulphuricum Dil..	,,	5	,,	20	0·296	,,	1·184	,,
,, Sulphurosum,	dr.	$\frac{1}{2}$ to dr.		1	1·776	,,	3·55	,,
,, Tartaricum,	gr.	5 to gr.		20	0·324	gm. to	1·296	,,
Aconitina,	,,	$\frac{1}{240}$	,,	$\frac{1}{60}$	0·00027	,,	0·00108	,,
Æther,	min.	10 to min.		60	0·592	c.c. to	3·55	c.c
,, Aceticus,	,,	20	,,	90	1·184	,,	5·33	,,
Aloes,	gr.	2 to gr.		5	0·13	gm. to	0·324	gm
Aloinum,	,,	$\frac{1}{2}$	,,	2	0·032	,,	0·13	,,
Ammonii Benzoas,	,,	5	,,	15	0·324	,,	0·972	,,
,, Bromidum,	,,	5	,,	30	0·324	,,	1·944	,,
,, Carbonas,	,,	3	,,	10	0·194	,,	0·648	,,
,, Chloridum,	,,	3	,,	20	0·194	,,	1·296	,,
,, Phosphas,	,,	5	,,	20	0·324	,,	1·296	,,
,, Salicylas,	,,	5	,,	30	0·324	,,	1·944	,,
Amyl Nitris (*mouth*),	min.	$\frac{1}{2}$ to min.		1	0·03	c.c. to	0·059	c.c
,, (*inhaled*).	,,	2	,,	5	0·118	,,	0·296	,,
Antifebrinum,	gr.	1 to gr.		3	0·065	gm. to	0·194	gm
Antimonii Oxidum,	,,	1	,,	2	0·065	,,	0·13	,,
Antimonium Sulphuratum,	,,	1	,,	2	0·065	,,	0·13	,,
,, Tartaratum (*diaphoretic*),	,,	$\frac{1}{24}$	,,	$\frac{1}{8}$	0·0027	,,	0·008	,,
,, ,, (*emetic*),	,,	1	,,	2	0·065	,,	0·13	,,
Antipyrinum,	,,	5	,,	20	0·324	,,	1·296	,,
Apomorphidæ Hydrochloridum,	,,	$\frac{1}{16}$	,,	$\frac{1}{4}$	0·004	,,	0·016	,,
Arsenii Iodidum,	,,	$\frac{1}{20}$	,,	$\frac{1}{8}$	0·0032	,,	0·013	,,
Asafetida,	,,	5	,,	15	0·324	,,	0·972	,,
Aspirine,	,,	5	,,	15	0·324	,,	0·972	,,
Atropina,	,,	$\frac{1}{200}$	,,	$\frac{1}{100}$	0·00032	,,	0·00065	,,
Atropinæ Sulphas,	,,	$\frac{1}{200}$	,,	$\frac{1}{100}$	0·00032	,,	0·00065	,,
Bismuthi Carbonas.	,,	5	,,	20	0·324	,,	1·296	,,
,, Oxidum,	,,	5	,,	20	0·324	,,	1·296	,,
,, Salicylas,	,,	5	,,	20	0·324	,,	1·296	,,
,, Subgallas,	,,	10	,,	20	0·648	,,	1·296	,,
,, Subnitras,	,,	5	,,	20	0·324	,,	1·296	,,
Borax,	,,	5	,,	20	0·324	,,	1·296	,,

	IMPERIAL.				METRIC.				
Bromoformum,	min.	½	to min.	2	0·03	c.c.	to	0·118	c.c.
Caffeina,	gr.	½	to gr.	4	0·032	gm.	to	0·259	gm.
Caffeinæ Citras,	,,	2	,,	10	0·13	,,		0·648	,,
,, ,, Efferves.	,,	60	,,	120	3·89	,,		7·78	,,
Calcii Chloridum,	,,	5	,,	15	0·324	,,		0·972	,,
,, Hypophosphis,	,,	3	,,	10	0·194	,,		0·648	,,
,, Phosphas,	,,	5	,,	15	0·324	,,		0·972	,,
,, Sulphidum,	,,	¼	,,	1	0·016	,,		0·065	,,
Calomel,	,,	1/10	,,	5	0·0065	,,		0·324	,,
Camphora,	,,	2	,,	5	0·13	,,		0·324	,,
Carbo Ligni,	,,	60	,,	120	3·89	,,		7·78	,,
Cerii Oxalas,	,,	2	,,	10	0·13	,,		0·648	,,
Chloral Hydras,	,,	5	,,	20	0·324	,,		1·296	,,
Chloroformum,	min.	1	to min.	5	0·059	c.c.	to	0·296	c.c.
Cocainæ Hydrochloridum,	gr.	1/5	to gr.	½	0·013	gm.	to	0·032	gm.
Codeina,	,,	¼	,,	2	0·016	,,		0·13	,,
Codeinæ Phosphas,	,,	¼	,,	2	0·016	,,		0·13	,,
Creosotum,	min.	1	to min.	5	0·059	c.c.	to	0·296	c.c.
Cubebæ Fructus,	gr.	30	to gr.	60	1·944	gm.	to	3·89	gm.
Cupri Sulphas (*astringent*),	,,	¼	,,	2	0·016	,,		0·13	,,
,, (*emetic*),	,,	5	,,	10	0·324	,,		0·648	,,
Elaterium,	,,	1/10	,,	½	0·0065	,,		0·032	,,
Ergota,	,,	20	,,	60	1·296	,,		3·89	,,
Ergotininæ Citras,	,,	1/200	,,	1/50	0·00032	,,		0·0013	,,
Ergotinum,	,,	2	,,	8	0·13	,,		0·518	,,
Eucalypti Gummi,	,,	2	,,	5	0·13	,,		0·324	,,
Euonyminum,	,,	1	,,	2	0·065	,,		0·13	,,
Ext. Aloes Barb.,	,,	1	,,	4	0·065	,,		0·259	,,
,, Belladonnæ Alcoholic,	,,	¼	,,	1	0·016	,,		0·065	,,
,, ,, Viride,	,,	¼	,,	1	0·016	,,		0·065	,,
Cannabis Indicæ,	,,	¼	,,	1	0·016	,,		0·065	,,
,, Cascaræ Sagradæ,	,,	2	,,	8	0·13	,,		0·518	,,
,, ,, Liq.,	,,	½	,,	1	1·776	c.c.	to	3·55	c.c.
,, Ipecacuanhæ Liq. (*expect.*),	min.	½	to min.	2	0·03	,,		0·118	,,
,, ,, (*emetic*),	,,	15	,,	20	0·888	,,		1·184	,,
,, Jalapæ,	gr.	2	to gr.	8	0·13	gm.	to	0·518	gm.
,, Nucis Vomicæ,	,,	¼	,,	1	0·016	,,		0·065	,,
,, ,, Liq.,	min.	1	to min.	3	0·059	c.c.	to	0·178	c.c.
,, Opii,	gr.	¼	to gr.	1	0·016	gm.	to	0·06	gm.
,, ,, Liq.,	min.	5	to min.	30	0·296	c.c.	to	1·776	c.c.
,, Physostigmatis,	gr.	¼	to gr.	1	0·016	gm.	to	0·065	gm.
,, Strophanthi,	,,	¼	,,	1	0·016	,,		0·065	,,
Ferri Arsenas,	,,	1/16	,,	¼	0·004	,,		0·016	,,
,, Carbonas Saccharatus,	,,	10	,,	30	0·648	,,		1·944	,,
,, et Ammonii Citras,	,,	5	,,	10	0·324	,,		0·648	,,
,, et Quininæ Citras,	,,	5	,,	10	0·324	,,		0·648	,,
,, Iodidum,	,,	1	,,	5	0·065	,,		0·334	,,
,, Phosphas,	,,	5	,,	10	0·324	,,		0·648	,,
,, et Quininæ et Strychninæ Citras,	,,	3	,,	6	0·194	,,		0·309	,,
,, Sulphas,	,,	1	,,	5	0·065	,,		0·334	,,
Ferrum Redactum,	,,	1	,,	5	0·065	,,		0·324	,,
,, Tartaratum,	,,	5	,,	10	0·324	,,		0·648	,,
Glycerinum Pepsini,	dr.	1	to dr.	2	3·55	c.c.	to	7·1	c.c.
Guaiaci Resina,	gr.	5	to gr.	15	0·324	gm.	to	0·972	gm.
Guaiacol,	min.	1	to min.	5	0·059	c.c.	to	0·296	c.c.
,, Carbonas,	gr.	5	to gr.	10	0·324	gm.	to	0·648	gm.
Homatropinæ Hydrobromidum,	,,	1/80	,,	1/20	0·00081	,,		0·0032	,,
,, Hydrochloridum,	,,	1/80	,,	1/20	0·00081	,,		0·0032	,,
Hydrarg. Iodidum Flavum,	,,	⅛	,,	1	0·008	,,		0·065	,,
,, ,, Rubrum,	,,	1/32	,,	1/16	0·002	,,		0·004	,,

	IMPERIAL.		METRIC.	
*Hydrarg. Iodidum Viride,	gr. $\frac{1}{8}$	to gr. 1	0·008	gm. to 0·065 gm.
,, Perchloridum,	,, $\frac{1}{32}$	,, $\frac{1}{16}$	0·002	,, 0·004 ,,
,, Subchloridum,	,, $\frac{1}{10}$	,, 5	0·0065	,, 0·324 ,,
,, c̄ Cretâ,	,, $\frac{1}{4}$	,, 5	0·016	,, 0·324 ,,
Hyoscinæ Hydrobromidum,	,, $\frac{1}{200}$	,, $\frac{1}{100}$	0·00032	,, 0·00065 ,,
Hyoscyaminæ Sulphas,	,, $\frac{1}{200}$	,, $\frac{1}{100}$	0·00032	,, 0·00065 ,,
*Ichthyol,	,, $2\frac{1}{2}$	,, 10	0·162	,, 0·648 ,,
Ipecacuanhæ Rad. (*expect.*),	,, $\frac{1}{4}$	,, 2	0·016	,, 0·13 ,,
,, (*emetic*),	,, 15	,, 30	0·972	,, 1·944 ,,
Jalapa,	,, 5	,, 20	0·324	,, 1·296 ,,
Jalapæ Resina,	,, 2	,, 5	0·13	,, 0·324 ,,
*Jalapinum,	,, 1	,, 5	0·065	,, 0·324 ,,
Liquor Arsenicalis (Fowler),	min. 2	to min. 8	0·118	c.c. to 0·474 c.c.
,, Arsenici Hydrochlor.,	,, 2	,, 8	0·118	,, 0·474 ,,
,, Arsenici et Hydrarg. Iodidi (Donovan),	,, 5	,, 20	0·296	,, 1·184 ,,
,, Bism. et Ammon. Cit.,	dr. $\frac{1}{2}$	to dr. 1	1·776	,, 3·55 ,,
,, Calumbæ Conc.,	,, $\frac{1}{2}$	,, 1	1·776	,, 3·55 ,,
,, Ferri Perchloridi,	min. 5	to min. 15	0·296	,, 0·888 ,,
,, ,, Pernitratis,	,, 5	,, 15	0·296	,, 0·888 ,,
,, Hydrarg. Perchlor.,	dr. $\frac{1}{2}$	to dr. 1	1·776	,, 3·55 ,,
,, Hydrogenii Peroxidi,	,, $\frac{1}{2}$	,, 2	1·776	,, 7·1 ,,
,, Morphinæ Acetatis,	min. 10	to min. 60	0·592	,, 3·55 ,,
,, ,, Hydrochloridi,	,, 10	,, 60	0·592	,, 3·55 ,,
,, ,, Tartratis,	,, 10	,, 60	0·592	,, 3·55 ,,
,, Potassæ,	,, 10	,, 30	0·592	,, 1·776 ,,
,, Quassiæ Conc.,	dr. $\frac{1}{2}$	to dr. 1	1·776	,, 3·55 ,,
,, Rhei Conc.,	,, $\frac{1}{2}$	,, 1	1·776	,, 3·55 ,,
,, Sarsæ Comp. Conc.,	,, 2	,, 8	7·1	,, 28·42 ,,
,, Senegæ Conc.,	,, $\frac{1}{2}$	,, 1	1·776	,, 3·55 ,,
,, Sennæ Conc.,	,, $\frac{1}{2}$	,, 1	1·776	,, 3·55 ,,
,, Serpentariæ Conc.,	,, $\frac{1}{2}$	,, 2	1·776	,, 7·1 ,,
,, Sodii Arsenatis,	min. 2	to min. 8	0·118	,, 0·474 ,,
,, Strychninæ Hydrochlor.,	,, 2	,, 8	0·118	,, 0·474 ,,
,, Thyroidei,	,, 5	,, 15	0·296	,, 0·888 ,,
Lithii Carbonas,	gr. 2	to gr. 5	0·13	gm. to 0·324 gm.
,, Citras,	,, 5	,, 10	0·324	,, 0·648 ,,
,, ,, Effervescens,	,, 60	,, 120	3·89	,, 7·78 ,,
Magnesii Carbonas,	,, 5	,, 60	0·324	,, 3·89 ,,
,, Sulphas,	,, 30	to oz. $\frac{1}{2}$	1·944	,, 14·17 ,,
,, ,, Effervescens,	dr. 1	,, 1	3·89	,, 28·35 ,,
* ,, Sulphis,	gr. 10	to gr. 30	0·648	,, 1·944 ,,
*Manganesii Dioxidum,	,, 2	,, 10	0·13	,, 0·648 ,,
Menthol,	,, $\frac{1}{2}$	,, 2	0·032	,, 0·13 ,,
*Mercuric Potassium Iodide,	,, $\frac{1}{12}$	,, $\frac{1}{3}$	0·0054	,, 0·022 ,,
*Methylene Blue,	,, 1	,, 4	0·065	,, 0·259 ,,
*Morphina,	,, $\frac{1}{10}$	,, $\frac{1}{2}$	0·0065	,, 0·032 ,,
Morphinæ Acetas,	,, $\frac{1}{8}$	,, $\frac{1}{2}$	0·008	,, 0·032 ,,
,, Hydrochloridum,	,, $\frac{1}{8}$	,, $\frac{1}{2}$	0·008	,, 0·032 ,,
* ,, Phosphas,	,, $\frac{1}{8}$	,, $\frac{1}{2}$	0·008	,, 0·032 ,,
* ,, Sulphas,	,, $\frac{1}{8}$	,, $\frac{1}{2}$	0·008	,, 0·032 ,,
,, Tartras,	,, $\frac{1}{8}$	,, $\frac{1}{2}$	0·008	,, 0·032 ,,
Naphthol β,	,, 3	,, 10	0·194	,, 0·648 ,,
Nitroglycerinum (Trinitrinum),	,, $\frac{1}{200}$	,, $\frac{1}{100}$	0·00032	,, 0·0013 ,,
Oleum Cajuputi,	min. $\frac{1}{2}$	to min. 3	0·03	c.c. to 0·178 c.c.
,, Crotonis,	,, $\frac{1}{2}$	,, 1	0·03	,, 0·059 ,,
,, Eucalypti,	,, $\frac{1}{2}$	,, 3	0·03	,, 0·178 ,,
,, Phosphoratum,	,, 1	,, 5	0·059	,, 0·296 ,,
,, Santali,	,, 5	,, 30	0·296	,, 1·776 ,,
,, Terebinthinæ,	,, 2	,, 10	0·118	,, 0·592 ,,
,, ,, (*Anthelmintic*),	dr. 3	to dr. 4	10·65	,, 14·21 ,,

	IMPERIAL.					METRIC.		
Opium, - - - - - - -	gr.	½	to gr.	2	0·032	gm. to	0·13	gm.
Paraldehydum, - -	dr.	½	to dr.	2	1·776	c.c. to	7·1	c.c.
*Pelletierinæ Tannas, - - -	gr.	2	to gr.	8	0·13	gm. to	0·518	gm.
Phenacetinum, - - - - -	,,	5	,,	10	0·324	,,	0·648	,,
Phenazonum (Antipyrine), -	,,	5	,,	20	0·324	,,	1·296	,,
Phenol, - - - - - - -	,,	1	,,	3	0·065	,,	0·194	,,
Phosphorus, - - - - - -	,,	1/100	,,	1/20	0·00065	,,	0·0032	,,
*Physostigminæ Hydrobrom., -	,,	1/60	,,	1/20	0·00108	,,	0·0032	,,
* ,, Salicylas, - -	,,	1/60	,,	1/20	0·00108	,,	0·0032	,,
,, Sulphas, - -	,,	1/60	,,	1/20	0·00108	,,	0·0032	,,
Picrotoxinum, - - - - -	,,	1/100	,,	1/25	0·00065	,,	0·0026	,,
*Pilocarpinæ Hydrochloridum, -	,,	1/20	,,	1/3	0·0032	,,	0·022	,,
,, Nitras, - - - -	,,	1/20	,,	½	0·0032	,,	0·032	,,
Pil. Aloes Barbadensis, - -	,,	4	,,	8	0·259	,,	0·518	,,
,, ,, et Asafetidæ, - -	,,	4	,,	8	0·259	,,	0·518	,,
,, ,, et Ferri, -	,,	4	,,	8	0·259	,,	0·518	,,
,, ,, et Myrrhæ, - -	,,	4	,,	8	0·259	,,	0·518	,,
,, ,, Socotrinæ, - - -	,,	4	,,	8	0·259	,,	0·518	,,
,, Cambogiæ Comp., - - -	,,	4	,,	8	0·259	,,	0·518	,,
,, Colocynthidis Comp., - -	,,	4	,,	8	0·259	,,	0·518	,,
,, ,, et Hyoscyami,	,,	4	,,	8	0·259	,,	0·518	,,
,, Ferri, - - - - - -	,,	5	,,	15	0·324	,,	0·972	,,
,, Hydrargyri, - - - - -	,,	4	,,	8	0·259	,,	0·518	,,
,, Hydrarg. Subchlor. Co., -	,,	4	,,	8	0·259	,,	0·518	,,
,, Ipecac. c̄ Scillâ, - - -	,,	4	,,	8	0·259	,,	0·518	,,
,, Phosphori, - - - -	,,	1	,,	2	0·065	,,	0·13	,,
,, Plumbi c̄ Opio, - - -	,,	2	,,	4	0·13	,,	0·259	,,
,, Quininæ Sulphatis, - - -	,,	2	,,	8	0·13	,,	0·518	,,
,, Rhei Comp., - - - - -	,,	4	,,	8	0·259	,,	0·518	,,
,, Saponis Comp., - - - -	,,	2	,,	4	0·13	,,	0·259	,,
,, Scammonii Comp., - - -	,,	4	,,	8	0·259	,,	0·518	,,
,, Scillæ Comp., - - - -	,,	4	,,	8	0·259	,,	0·518	,,
Plumbi Acetas, - - - - -	,,	1	,,	5	0·065	,,	0·324	,,
Podophylli Resina, - - -	,,	¼	,,	1	0·016	,,	0·065	,,
Potassii Acetas, - - - - -	,,	10	,,	60	0·648	,,	3·89	,,
,, Bicarbonas, - - - -	,,	5	,,	30	0·324	,,	1·944	,,
,, Bichromas, - - - -	,,	1/10	,,	1/5	0·0065	,,	0·013	,,
,, Bromidum, - - - -	,,	5	,,	30	0·324	,,	1·944	,,
,, Carbonas, - - - -	,,	5	,,	20	0·324	,,	1·296	,,
,, Chloras, - - - - -	,,	5	,,	15	0·324	,,	0·972	,,
,, Citras, - - - - -	,,	10	,,	40	0·648	,,	2·592	,,
,, Iodidum, - - - - -	,,	5	,,	20	0·324	,,	1·296	,,
,, Nitras, - - - - -	,,	5	,,	20	0·324	,,	1·296	,,
,, Permanganas, - - -	,,	1	,,	3	0·065	,,	0·194	,,
,, Tartras, - - - - -	,,	30	,,	240	1·944	,,	15·55	,,
,, ,, Acidus, - - -	,,	20	,,	60	1·296	,,	3·89	,,
Pulvis Antimonialis, - - - -	,,	3	,,	6	0·194	,,	0·389	,,
,, Catechu Comp., - - -	,,	10	,,	40	0·648	,,	2·592	,,
,, Cinnamomi Comp., - -	,,	10	,,	40	0·648	,,	2·592	,,
,, Cretæ Aromaticus, - -	,,	10	,,	60	0·648	,,	3·89	,,
,, ,, c̄ Opio,	,,	10	,,	40	0·648	,,	2·592	,,
,, Ipecacuanhæ Comp., - -	,,	5	,,	15	0·324	,,	0·972	,,
,, Jalapæ Comp., - - - -	,,	20	,,	60	1·296	,,	3·89	,,
,, Kino Comp., - - - -	,,	5	,,	20	0·324	,,	1·296	,,
,, Opii Comp., - - - -	,,	2	,,	10	0·13	,,	0·640	,,
,, Rhei Comp., - - - -	,,	20	,,	60	1·296	,,	3·89	,,
,, Scammonii Comp., - - -	,,	10	,,	20	0·648	,,	1·296	,,
*Quinina, - - - - - - -	,,	1	,,	4	0·065	,,	0·259	,,
*Quininæ Bisulphas, - - - -	,,	1	,,	10	0·065	,,	0·648	,,
* ,, Hydrobromidum, - -	,,	1	,,	10	0·065	,,	0·648	,,
,, Hydrochloridum, - -	,,	1	,,	10	0·065	,,	0·648	,,

		IMPERIAL.			METRIC.			
Quininæ Hydrochlor. Acid,	gr.	1	to gr.	10	0·065	gm. to	0·648	gm
,, Salicylas,	,,	2	,,	6	0·13	,,	0·389	,,
,, Sulphas,	,,	1	,,	10	0·065	,,	0·648	,,
Resorcinum,	,,	3	,,	6	0·194	,,	0·389	,,
Rhei Radix,	,,	3	,,	30	0·194	,,	1·944	,,
Salicinum,	,,	5	,,	20	0·324	,,	1·296	,,
Salol,	,,	5	,,	15	0·324	,,	0·972	,,
Santoninum,	,,	2	,,	5	0·13	,,	0·324	,,
Scammoniæ Resina,	,,	3	,,	8	0·194	,,	0·518	,,
Soda Tartarata,	dr.	2	to dr.	4	7·78	,,	15·55	,,
Sodii Arsenas,	gr.	$\frac{1}{40}$	to gr.	$\frac{1}{10}$	0·0016	,,	0·0065	,,
,, Benzoas,	,,	5	,,	30	0·324	,,	1·944	,,
,, Bicarbonas,	,,	5	,,	30	0·324	,,	1·944	,,
,, Bromidum,	,,	5	,,	30	0·324	,,	1·944	,,
,, Hypophosphis,	,,	3	,,	10	0·194	,,	0·648	,,
,, Iodidum,	,,	5	,,	20	0·324	,,	1·296	,,
,, Phosphas,	,,	30	to oz.	$\frac{1}{2}$	1·944	,,	14·17	,,
,, ,, Effervescens,	dr.	1	to dr.	4	3·89	,,	15·55	,,
,, Salicylas,	gr.	10	to gr.	30	0·648	,,	1·944	,,
,, Sulphas,	,,	30	to oz.	$\frac{1}{2}$	1·944	,,	14·17	,,
,, ,, Effervescens,	dr.	1	to dr.	4	3·89	,,	15·55	,,
,, Sulphis,	gr.	5	to gr.	20	0·324	,,	1·296	,,
Spiritus Ætheris Comp.,	min.	20	to min.	90	1·184	c.c. to	5·33	c.c.
,, ,, Nitrosi,	,,	20	,,	90	1·184	,,	5·33	,,
,, Ammon. Arom.,	,,	20	,,	90	1·184	,,	5·33	,,
,, ,, Fetidus,	,,	20	,,	90	1·184	,,	5·33	,,
,, Camphoræ,	,,	5	,,	20	0·296	,,	1·184	,,
Strychnina,	gr.	$\frac{1}{60}$	to gr.	$\frac{1}{15}$	0·00108	gm. to	0·0043	gm
Strychninæ Hydrochloridum,	,,	$\frac{1}{60}$	,,	$\frac{1}{15}$	0·00108	,,	0·0043	,,
,, Nitras,	,,	$\frac{1}{60}$	,,	$\frac{1}{15}$	0·00108	,,	0·0043	,,
,, Sulphas,	,,	$\frac{1}{60}$	,,	$\frac{1}{15}$	0·00108	,,	0·0043	,,
Sulphonal,	,,	10	,,	30	0·648	,,	1·944	,,
Syrupus Calcii Lactophosph.,	dr.	$\frac{1}{2}$	to dr.	1	1·776	c.c. to	3·55	c.c.
,, Cascaræ Aromat.,	,,	$\frac{1}{2}$	,,	2	1·776	,,	7·1	,,
,, Chloral,	,,	$\frac{1}{2}$	,,	2	1·776	,,	7·1	,,
,, Codeinæ,	,,	$\frac{1}{2}$	,,	2	1·776	,,	7·1	,,
,, Ferri Phosph. c̄ Quininâ et Strychninâ (Easton)	,,	$\frac{1}{2}$	,,	1	1·776	,,	3·55	,,
,, Ferri Phosphatis,	,,	$\frac{1}{2}$	,,	1	1·776	,,	3·55	,,
,, ,, Comp.,	,,	$\frac{1}{2}$	,,	2	1·776	,,	7·1	,,
Terebenum,	min.	5	to min.	15	0·296	,,	0·888	,,
Thymol,	gr.	$\frac{1}{2}$	to gr.	2	0·032	gm. to	9·13	,,
Tinctura Aconiti,	min.	5	to min.	15	0·296	c.c. to	0·888	c.c.
,, ,, (repeated),	,,	2	,,	5	0·118	,,	0·296	,,
,, Aloes,	dr.	$\frac{1}{2}$	to dr.	2	1·776	,,	7·1	,,
,, Belladonnæ,	min.	5	to min.	15	0·296	,,	0·888	,,
,, Camphoræ Comp.,	dr.	$\frac{1}{2}$	to dr.	1	1·776	,,	3·55	,,
,, Cannabis Indicæ,	min.	5	to min.	15	0·296	,,	0·888	,,
,, Cantharidis,	,,	2	,,	15	0·118	,,	0·888	,,
,, Capsici,	,,	1	,,	15	0·059	,,	0·888	,,
,, Chlorof. et Morphinæ Comp.,	,,	5	,,	15	0·296	,,	0·888	,,
,, Cinchonæ Comp.,	dr.	$\frac{1}{2}$	to dr.	1	1·776	,,	3·55	,,
,, Colchici Seminum,	min.	5	to min.	15	0·296	,,	0·888	,,
,, Digitalis,	,,	5	,,	15	0·296	,,	0·888	,,
,, Ergotæ Ammon.,	dr.	$\frac{1}{2}$	to dr.	1	1·776	,,	3·55	,,
,, Ferri Perchloridi,	min.	5	to min.	15	0·296	,,	0·888	,,
,, Gelsemii,	,,	5	,,	15	0·296	,,	0·888	,,
,, Guaiaci Ammon.,	dr.	$\frac{1}{2}$	to dr.	1	1·776	,,	3·55	,,
,, Hamamelidis,	,,	$\frac{1}{2}$	,,	1	1·776	,,	3·55	,,
,, Hyoscyami,	,,	$\frac{1}{2}$	,,	1	1·776	,,	3·55	,,

	IMPERIAL.	METRIC.
Tinctura Iodi, - - - -	min. 2 to min. 5	0·118 c.c. to 0·296 c.c.
,, Jaborandi, - -	dr. ½ to dr. 1	1·776 ,, 3·55 ,,
,, Nucis Vomicæ, -	min. 5 to min. 15	0·296 ,, 0·888 ,,
,, Opii, - - - -	,, 5 ,, 30	0·296 ,, 1·776 ,,
,, ,, Ammoniata, -	dr. ½ to dr. 1	1·776 ,, 3·55 ,,
,, Podophylli, - -	min. 5 to min. 15	0·296 ,, 0·888 ,,
,, Pruni Virginianæ,	dr. ½ to dr. 1	1·776 ,, 3·55 ,,
,, Quininæ, - - -	,, ½ ,, 1	1·776 ,, 3·55 ,,
,, ,, Ammon., -	,, ½ ,, 1	1·776 ,, 3·55 ,,
,, Scillæ, - - - -	min. 5 to min. 15	0·296 ,, 0·888 ,,
,, Stramonii, - - -	,, 5 ,, 15	0·296 ,, 0·888 ,,
,, Strophanthi, - -	,, 5 ,, 15	0·296 ,, 0·888 ,,
,, Valerianæ Ammon.,	dr. ½ to dr. 1	1·776 ,, 3·55 ,,
,, Zingiberis, - -	,, ½ ,, 1	1·776 ,, 3·55 ,,
Trinitrin (Nitroglycerin), -	gr. 1/200 to gr. 1/50	0·00032 gm. to 0·0013 gm.
Trional, - - - - - -	,, 10 ,, 30	0·6 ,, 1·94 ,,
Veronal, - - - - - -	,, 5 ,, 10	0·324 ,, 0·648 ,,
Vinum Antimoniale (*diaphoretic*),	min. 10 to min. 30	0·592 c.c. to 1·776 c.c.
,, ,, (*emetic*),	dr. 2 to dr. 4	7·1 ,, 14·21 ,,
,, Colchici, - - -	min. 10 to min. 30	0·592 ,, 1·776 ,,
,, Ipecac. (*expectorant*),	,, 10 ,, 30	0·592 ,, 1·776 ,,
,, ,, (*emetic*), -	dr. 4 to dr. 6	14·21 ,, 21·31 ,,
Zinci Acetas, - - - -	gr. 1 to gr. 2	0·065 gm. to 0·13 gm.
,, Oxidum, - - - -	,, 2 ,, 10	0·13 ,, 0·648 ,,
,, Sulphas (*tonic*), - -	,, 1 ,, 3	0·065 ,, 0·194 ,,
,, ,, (*emetic*), - -	,, 10 ,, 30	0·648 ,, 1·944 ,,

APPENDIX V.

TROPICAL ASPECTS OF EXAMINATION FOR LIFE ASSURANCE.

By D. J. Galloway, M.D., F.R.C.P.

LIFE ASSURANCE.

THERE are certain aspects in which life assurance in the tropics differs vitally from the home standard, and in the following short appendix it is not proposed to do more than indicate some of those divergencies briefly and suggestively.

European Lives.—The European who migrates to the tropics with a view to becoming resident there is, more often than otherwise, a selected life. *First*, the grade of society from which he is drawn is usually that of the professional or upper mercantile classes, and he has had the advantage of a public school, gymnasium, lycee or university education. In any case his circumstances and education have been those most conducive to the development of a sound body; and *secondly*, the exigencies of work in the tropics are such that every care is taken to obtain the highest value in every working unit, and thus the individual or firm to whom he goes out insist on a most rigorous examination as to physical fitness. It is also common, in Continental races, to have a record of some years military service.

Many offices insist on a small added percentage during the first five years of the currency of a tropical policy, while others discard the extra premium altogether. The wise course is a middle one—*i.e.*, to penalise the first five years of tropical residence. This will commend itself to all who have been much in contact with newcomers to the tropics, as none know better than they, that the risks, whether physical or moral (with material physical results from an insurance standpoint), are greatest during that period. But, given the usual "selected" life which has won through this probationary period without deterioration, all rational grounds for an added percentage are gone.

Native Lives.—Natives of a good class are alive to the advantages of life assurance. Life policies could be issued only after the establishment of a life rate, and that again is dependent on the expectation of the individual. The observation of a number of races during many years goes far to establish the presumption that the expectation of life in different ethnic groups must vary widely, and, so far, this is the only kind of evidence obtainable, the statistics necessary, as the basis of life tables, being either non-existent or incomplete. Life risks are, therefore, for the present, best avoided; but a safe and profitable business may be done in term policies practically at the same rates as for Europeans. The difficulty in examining native lives lies in the impossibility of obtaining a reliable family history. This is most accentuated in polygamic races. Even the personal history is mostly a matter of conjecture. Thus, deprived of such valuable sources of information, the examiner has to form his estimate of the value of the life under consideration solely upon the result of his personal examination, which has, of necessity, to be exceedingly thorough and minute.

The Influence of Disease-factors—1. *Obesity.*—Of the many factors which may conduce to the production and deposition of an abnormal amount of adipose tissue in the tropical resident the acquisition of a habit of eating and drinking to excess is foremost. The drink need not of necessity be alcoholic, though, if so, the effects as to production of fat are enhanced. To prevent misconception it may be well to discuss here the question of excessive weight. It falls to the lot of every examiner to have to consider the case of men who are of powerful build, and in whom the weight ratio is out of all proportion to the height. They are, as a rule, most desirable lives, and, as often as not, their papers are returned by the scrutineers queried or absolutely refused on the ground of excessive weight. A reference to the relation which the abdominal girth bore to the chest measurement might have saved the situation.

The limit at which the accumulation of fat ceases to be physiological, and becomes a menace to health or life, must, of necessity, be left to the judgment of the individual examiner, and thus is somewhat arbitrary. The bearing which obesity has to life assurance may be summarised as follows :—

It may, especially in some native races, be associated with Glycosuria, which may be continuous or intermittent. The danger of passing a case during an intermission is obvious.

It may be, and generally is, associated with a degree of arteriosclerosis, and, frequently, an intermittent albuminuria.

Obese persons develop heat more readily and store it up more than normal individuals. The symptoms of cardiac distress evoked by any rise of atmospheric temperature and the dangers of heat apoplexy after any unwonted exertion are matters of common knowledge. But a more real danger is that of contracting any of the many febrile diseases of the tropics. The tendency to hyperpyrexia is always very great, and, should such a contingency occur, the benefit to be obtained from hydrotherapy is reduced to the vanishing point.

2. *Malaria.*—The factor which introduces into tropical life assurance its most speculative element is malaria ; nor does this seem capable of elimination by any amount of care on the part of the examiner. The more closely he is in touch with his district the more clearly is it demonstrated to him that from year to year in any given locality, while adhering to its original type, malaria may fluctuate in its prevalence or in its severity : or, indeed, the type itself may change. It is not customary to lien policies in a country in which the simple tertian is the endemic type, but it may cogently be argued that the assurance companies should receive the benefit of the doubt in view of any change to a more malignant type. This benefit they, indirectly though very practically, have received in the spread during recent years of the knowledge of the relationship between surface puddles, mosquitoes and malaria, and the energetic "mosquito war" waged wherever the white man chooses to settle.

It is otherwise, however, in those instances in which the applicant proposes to reside in a malarial district notorious either for the prevalence or malignity of its type. With these may be classed those not uncommon proposals from persons about to explore or open up little-known localities (pioneer risks). The extra rate to be applied should not be based on the virulence of the type of malaria alone, but should take into consideration as an item of at least equal value the distance, in hours, at which the proposed is likely to be situated from skilled assistance in the event of illness.

It is usual to remit the extra impost when the person leaves the locality on account of which it was imposed, though some offices insist on its continuance until he leaves the tropics.

Under the term *Malarial Cachexia* is grouped a most heterogeneous array, from cases with livid icy extremities and without splenic tumour, in which nature seems to have offered no resistance to the intoxication, to those in whom a moderate degree of splenic enlargement is the only departure from the normal. With this latter exception the whole class is uninsurable so long as they are resident in the tropics, and it is doubtful if those algid cases, or, indeed, any which show marked nerve implication, will ever, under any circumstances, become eligible lives. In considering the latter-mentioned class it is advisable not to put too much weight on a moderate degree of splenic tumour, as a condition of easy palpability may persist throughout life, and yet not be incompatible with perfect health.

3. *The Liver.*—The active hyperæmia with which the liver of the tropical newcomer is credited is one of the risks which legitimately fall to be covered by the added premium for the first five years of tropical residence. The free living, which is assumed to be the cause of this hyperæmia, is not usually of long duration. "Wisdom comes with years," or, more correctly, wisdom assisted by sundry digestive crises comes with months. Should this desirable climax not be attained and the engorgement become permanent, it is certain to be accompanied by other departures from the normal of such a nature as to render the task of assessing the extra risk a comparatively easy one. Thus, singly, or in various combination, obesity, arteriosclerosis, albuminuria, cardiac changes, and signs of a chronic auto-intoxication will have to be reckoned with.

In discussing the liver of malaria there is a danger of confounding two types—one grave, the other comparatively innocuous. The former is that described in most text books as occurring in the cachectic stage, and is associated with a much enlarged spleen and profound blood changes. The other type of liver enlargement is mostly found in young people who have been in a malarious district for a few months and who have not taken quinine. It is very massive, is associated with but little splenic enlargement, and not at all with cachexia. The history given is that they never have fever unless they leave their own district. The effect of the migration is to induce a series of typical malarial attacks, aided probably by the

free administration of quinine which these render necessary, and a speedy reduction of the size of the liver to normal. The absence of cachexia would seem to indicate this condition to be a stage in the working out of an immunity which, unfortunately, is merely local, and it merits notice only that it may not be confused with the grave form already mentioned.

It occasionally happens that a case with a history of *antecedent liver abscess* presents itself for examination. If it has been one of an acute frank solitary abscess with a rapid and complete convalescence after evacuation, followed by an interval of two years of perfect health, both as to liver and bowel, there can be no cavil as to its acceptance as a good risk.

The liver to be avoided as undesirable is that where, in the course of ordinary examination, the hepatic region is seen to bear marks of having at some time or other received much attention, evidenced by the staining of the cutis from prolonged poulticing, marks of blisters, cuppings, exploratory punctures, &c. The history given is usually one of repeated recurrences of what, for want of a better definition, may be styled "liver abscess without the abscess," with a prolonged and tedious convalescence, and, frequently, a close relation to intercurrent dysenteric outbreaks.

4. *Dysentery and Chronic Diarrhœa.*—The researches of the past few years have, by demonstrating the many organisms which, singly or in combination, enter into the etiology of dysentery, settled the old clinical problem of the intense virulence of some infections and the comparative innocuousness of others. While this is of interest it is of little practical value from the point of view of the examiner in comparison to the other point which those researches have brought out—that most chronic diarrhœas of the tropics are in reality chronic dysenteries, a reversion to the opinion expressed by French writers twenty years ago, and looked upon by them as a condition of great gravity.

5. *Sprue.*—Individuals affected with the type of intestinal disease known as sprue are totally uninsurable. There is probably no class of diseases on which a residence in a temperate climate produces such markedly beneficial effects as in the alvine fluxes, and much caution should be exercised in accepting such a life which has just returned from such a holiday optimistically regarded as cured. In such a case one would counsel postponement for at least a year before taking it even into consideration.

6. *Blackwater Fever.*—Insurances of individuals proceeding to a "blackwater" district are carried through at a multiple premium (four or five), and this rate holds until the person has returned to a temperate climate and been resident there for at least twelve months. It is unlikely that any standard rate will be arrived at until there has been a revision of the content of the term "blackwater fever," as at present it is used to cover such fevers as bilious, remittent, and such accompaniments of fevers as the hæmaturia of malarial nephritis.

Analysis of the term "Fever."—Very early in the list of questions to be answered by the proposed occurs that relative to previous illnesses, and in very many cases the answer returned to the examiner in the tropics is "Fever." At first sight it appears as if he would be put to the disagreeable necessity of having to add to the already lengthy catechism to which the candidate is subjected, but if he accepts the term for the moment and proceeds with the examination the matter simplifies itself as he proceeds. There is one class whose inherent qualities cause them to be otherwise than lightly remembered, and these do not form part of the question. This group comprises yellow fever, blackwater fever, and malignant malaria. The usual question as to "duration of illness" isolates another group, the fevers of long continuance being either typhoid or Mediterranean. Proceeding with the physical examination, those fevers which leave objective sequelæ would be set aside:—The trypanosome (splenomegaly), leprous (maculæ), and filarial (glands, elephantiasis, &c.) fevers.

There yet remain a number of fevers of short duration—dengue, influenza, relapsing fever, simple malaria, and some of the fevers of the colon group—to be dealt with, and a single query as to any prevailing epidemic still further narrows the field.

These fragmentary notes, although possibly not of a nature to add much to the knowledge of the tropical examiner, may serve to bring home to those most concerned (the insurance companies) the necessity which exists for the collection of statistical information which would place their exploitation of this almost illimitable field of enterprise on a more scientific basis. At present their success depends, much more than in temperate climes, on the acumen of their individual examiners.

APPENDIX VI.

COMPARISON OF FAHRENHEIT AND CENTIGRADE SCALES.

HOW TO MAKE PERCENTAGE SOLUTIONS.

RELATIVE HUMIDITY FROM WET AND DRY BULBS.

PERCENTAGE COMPOSITION OF VARIOUS ANIMAL MILKS.

Thermometers.

CENTIGRADE AND FAHRENHEIT.

To convert degrees F. into degrees C., deduct 32, multiply by 5, and divide by 9.

To convert degrees C. into degrees F., multiply by 9, divide by 5, and add 32.

F.	C.	F.	C.	F.	C.	F.	C.
212	100	101·5	38·6	96	35·6	80	26·7
200	93·3	101	38·3	95·5	35·3	78	25·6
150	65·6	100·5	38·1	95	35	76	24·4
112	44·4	100	37·8	94·5	34·7	74	23·3
110	43·3	99·5	37·5	94	34·4	72	22·2
108	42·2	99	37·2	92	33·3	70	21·1
106	41·1	98·5	36·9	90	32·2	68	20
105	40·6	98	36·7	88	31·1	66	18·9
104	40	97·5	36·4	86	30	64	17·8
103	39·4	97	36·1	84	28·9	62	16·7
102	38·9	96·5	35·8	82	27·8	60	15·6

The Metric System of Weights and Measures.

FACTORS FOR CONVERTING FROM ONE SCALE TO THE OTHER.

To convert grammes into grains	× 15·432.
,, ,, ,, ounces, avoir. . .	× 0·03527.
,, kilogrammes into pounds . . .	× 2·2046.
,, grains into grammes	× 0·0648.
,, avoir. ounces into grammes . . .	× 28·35.
,, troy ounces into grammes . . .	× 31·104.
,, cubic centimetres into fluid ounces imperial	× 0·0352.
,, litres into fluid ounces imperial . .	× 35·2.
,, fluid ounces into cubic centimetres .	× 28·42.
,, pints into litres	× 0·568.
,, metres into inches	× 39·37.
,, inches into metres	× 0·0254.

Average Weights and Measurements of Adult Human Organs.

Heart.—Weight, male, 10 ozs. to 12 ozs.; female, 8 ozs. to 10 ozs. Measurement, 5 inches long, $3\frac{1}{2}$ inches broad, $2\frac{1}{2}$ inches thick.

Lungs.—Weight, right, 23 ozs.; left, 19 ozs.: very variable.

Stomach.—Weight, $4\frac{1}{2}$ ozs. to 5 ozs. Measurement, 10 inches to 12 inches long, 4 inches to 5 inches wide.

Liver.—Weight, 45 ozs. to 60 ozs. Measurement, transverse, 10 inches to 12 inches; antero-posterior, 6 inches to 7 inches.

Pancreas.—Weight, 3 ozs. Measurement, 6 inches to 8 inches long, $1\frac{1}{2}$ inches broad.

Spleen.—Weight, 5 ozs. to 7 ozs. Measurement, 5 inches long, 3 inches broad, $1\frac{1}{2}$ inches thick.

Kidney.—Weight, $4\frac{1}{2}$ ozs. to $5\frac{1}{2}$ ozs. Measurement, 4 inches long, $2\frac{1}{2}$ inches broad, $1\frac{1}{4}$ inches thick.

Brain.—Weight, male, 50 ozs.; female, 44 ozs.

The average weight and size of all these organs is less in the female than in the male.

Table of Percentage Solutions.

QUANTITY OF MEDICAMENT REQUIRED FOR MAKING.

Percentage.	1 fluid drachm.	1 fluid ounce.	1 pint.	Parts.
1	gr. 0·547	gr. 4·375	gr. 87·5	1 in 100
2	gr. 1·094	gr. 8·750	gr. 175·0	1 in 50
3	gr. 1·640	gr. 13·125	gr. 262·5	1 in 33·33
4	gr. 2·187	gr. 17·500	gr. 350·0	1 in 25
5	gr. 2·734	gr. 21·875	gr. 437·5	1 in 20
6	gr. 3·281	gr. 26·250	gr. 525·0	1 in 16·66
7	gr. 3·828	gr. 30·625	gr. 612·5	1 in 14·28
8	gr. 4·375	gr. 35·000	gr. 700·0	1 in 12·5
9	gr. 4·922	gr. 39·375	gr. 787·5	1 in 11·11
10	gr. 5·468	gr. 43·750	gr. 875·0	1 in 10

The above table has been arranged on the basis of 87·5 grains of solid in 1 pint (approximately 1 grain in 110 minims) of solvent: this being the method officially recognised for making a 1 per cent. solution. The substance should be dissolved in about two-thirds of the total quantity of solvent, and the bulk finally adjusted to the prescribed amount.

RELATIVE HUMIDITY TABLES AT NORMAL PRESSURE.

Reading of Wet Bulb.	Difference of Reading between Wet and Dry Bulbs in Degrees Fahrenheit.														
	0	1	2	3	4	5	6	7	8	9	10	11	12	13	14
60	100	94	89	83	78	73	68	64	60	56	53	49	46	43	40
61	100	94	89	84	78	73	69	65	61	57	53	50	46	43	40
62	100	94	89	84	79	74	70	65	61	57	54	51	47	44	41
63	100	95	89	84	79	74	70	66	62	58	55	51	48	45	42
64	100	96	89	84	79	75	70	66	62	58	55	52	49	46	43
65	100	96	90	85	80	75	71	67	63	59	56	53	49	46	43
66	100	96	90	85	80	76	71	67	63	60	56	53	50	47	44
67	100	96	90	85	80	76	72	68	64	60	57	54	51	48	45
68	100	96	90	85	81	76	72	68	64	61	58	54	51	48	45
69	100	96	90	85	81	76	72	69	65	61	58	55	52	49	46
70	100	96	90	86	81	77	73	69	65	61	58	56	52	49	47
71	100	96	99	86	81	77	73	70	66	62	59	56	53	50	47
72	100	96	90	86	82	77	74	70	66	62	60	57	54	51	48
73	100	96	91	86	82	78	74	70	67	63	60	57	54	51	49
74	100	96	91	86	82	78	74	71	67	63	60	58	55	52	49
75	100	96	91	86	82	78	74	71	67	64	61	58	55	52	50
76	100	96	91	87	82	78	75	71	68	64	61	58	56	53	50
77	100	96	91	87	83	79	75	72	68	65	62	59	56	53	51
78	100	96	91	87	83	79	75	72	68	65	62	59	56	54	51
79	100	96	91	87	83	79	76	72	69	66	63	60	57	54	52
80	100	96	91	87	83	79	76	72	69	66	63	60	57	55	52
81	100	96	91	87	83	80	76	73	69	66	63	61	58	55	53
82	100	96	91	87	84	80	76	73	70	67	64	61	58	56	53
83	100	96	92	88	84	80	77	73	70	67	64	61	59	56	54
84	100	96	92	88	84	80	77	74	70	67	64	62	59	56	54
85	100	96	92	88	84	81	77	74	71	68	65	62	59	57	54
86	100	96	92	88	84	81	77	74	71	68	65	62	60	57	55
87	100	96	92	88	84	81	78	74	71	68	65	63	60	58	55
88	100	96	92	88	85	81	78	75	72	69	66	63	60	58	56
89	100	96	92	88	85	81	78	75	72	69	66	63	61	58	56

RELATIVE HUMIDITY TABLES—*Continued.*

Reading of Wet Bulb.	Difference of Reading between Wet and Dry Bulbs in Degrees Fahrenheit.															
	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30
60	37	34	31	29	27	25	23	21	19	17	15	14	7	6	5	3
61	38	35	32	30	28	26	24	22	20	18	16	15	8	7	6	4
62	38	36	33	31	29	26	25	23	21	19	17	16	9	8	7	5
63	39	37	34	32	30	27	26	23	22	20	18	17	10	9	8	6
64	40	37	35	33	30	28	26	24	23	21	19	18	11	10	9	7
65	41	38	36	33	31	29	27	25	23	22	20	19	12	11	9	8
66	42	39	36	34	32	30	28	26	24	23	21	19	13	12	10	9
67	42	40	37	35	33	31	29	27	25	23	22	20	14	13	11	10
68	43	40	38	36	34	32	30	28	26	24	23	21	15	14	12	11
69	44	41	39	36	34	32	30	28	27	25	23	22	16	15	13	12
70	44	42	39	37	35	33	31	29	27	26	24	23	17	16	14	13
71	45	42	40	38	36	34	32	30	28	[illegible]	25	23	18	17	15	14
72	45	43	41	38	36	34	32	31	29	27	26	24	19	17	16	15
73	46	44	41	39	37	35	33	31	29	28	26	25	20	18	17	16
74	47	44	42	40	38	36	34	32	30	29	27	25	21	19	18	16
75	47	45	43	40	38	36	34	33	31	29	28	26	21	20	19	17
76	48	45	43	41	39	37	35	33	31	30	28	27	22	21	19	18
77	48	46	44	42	40	38	36	34	32	30	29	27	23	21	20	19
78	49	47	44	42	40	38	37	34	33	31	30	28	23	22	21	19
79	49	47	45	43	41	39	37	35	33	32	30	29	24	22	21	20
80	50	47	45	43	41	39	38	36	34	32	31	29	24	23	22	21
81	50	48	46	44	42	40	38	36	34	33	31	30	25	24	23	21
82	51	48	46	44	42	40	39	37	35	33	32	30	26	25	23	22
83	51	49	47	45	43	41	39	37	36	34	32	31	26	25	24	23
84	52	49	47	45	43	41	40	38	36	35	33	31	27	26	25	23
85	52	50	48	46	44	42	40	38	37	35	33	32	28	26	25	24
86	52	50	48	46	44	42	41	39	37	36	34	33	28	27	26	25
87	53	51	49	47	45	43	42	39	38	36	34	33	29	28	26	25
88	53	51	49	47	45	43	42	40	38	36	35	34	29	28	27	26
89	54	52	49	47	46	44	42	40	39	37	35	34	30	29	27	26

Percentage Composition of various Milks.

	Sp. Gr.	Proteid.	Total Solids.	Fat.	Lactose.	Salts.	Water.
Mares', .	1,035	1·99	9·22	1·21	5·67	·35	90·78
Asses', .	1,023-35	2·22	10·36	1·64	5·99	·51	89·64
Human, .	1,027	2·29	12·59	3·78	6·21	·31	87·41
Cows' .	1,032	3·55	12·83	3·69*	4·88	·71	87·41
Goats' .	1,032	4·29	14·29	4·78	4·46	·76	85·71
Buffaloes',	1,032	6·10	18·59	7·47†	4·15	·87	81·41

* Out of 187,600 analyses of cows' milk the maximum fat was 4·1 and the minimum 2·7. A slightly higher result is sometimes found in the tropics.

† The high percentage of fat in buffaloes' milk will permit of much watering and still show a high fat result. This is well known and much practised by the crafty native milkseller in the many tropical regions where buffaloes abound.

APPENDIX VII.

TROPICAL INFECTIOUS DISEASES.

QUARANTINE TABLE OF TROPICAL INFECTIOUS DISEASES.

Disease.	Method of Transmission.	Incubation (in days).	Date of Definite Illness on which Eruption appears.	Date of Definite Illness on which Begins to fade.	Quarantine after Latest Exposure.	Infection Ceases.
Chicken-pox.	? Aerial. ? Direct corporeal. ? Indirect corporeal.	10 to 16	1st day	4th day	20 days *	When all scabs are gone.
Cholera.	*Direct*—Food, water, and soiled products introduced into the mouth. *Indirect*—Flies.	½ to 5 (average 1 to 2)	Nil	Nil	7 days * †	After stools are perfectly normal.
Dengue.	? *Stegomyia fasciata.*	1 to 5	*First* 1st day. *Second* 6th day.	2nd day and 7th day	7 days ‡	When temperature is finally normal.
Malaria.	*Anopheles.* ? Other methods.	2 to 5	Nil	Nil	7 days §	When malarial parasite has left the blood.
Measles.	Direct corporeal. Indirect corporeal.	10 to 14	4th day. (Highly infectious 2 days before rash.)	5th to 7th day	16 days *	Not less than 2 weeks from appearance of the rash.
Plague.	Innoculation through wounds, flea bites, &c. ? Direct inhalation.	1 to 10 (average 2 to 5)	Nil	Nil	12 days * ‖	Eight weeks from onset of the disease.
Small-pox.	? Aerial. ? Direct corporeal. ? Indirect corporeal.	12 to 14	3rd day	9th or 10th day	16 days *	When every scab has fallen off.
Yellow fever.	*Stegomyia fasciata.*	1 to 13 (average 2 to 5)	Nil	Nil	13 days ¶	On the 4th day of the fever.

(*Brooke.*)

* This quarantine for contacts, fixed at two days longer than the maximum incubation period, can only be considered safe if *thorough* disinfection of person and effects is carried out *at the commencement* of the quarantine period. A further disinfection at the end of that period is also advisable.

† In cholera quarantine, water and food supply should also be enquired into and dealt with. Moreover, the destruction of flies should receive attention.

‡ In such a benign disease as dengue, quarantine can be dispensed with.

§ Owing to the comparatively small number of malignant cases, quarantine is not imposed against malaria contacts, as in the somewhat analogous yellow fever. The mosquito is not infectious until 12 or 14 days after feeding on infected blood. See remarks below on yellow fever.

‖ Attention should also be directed to the possibility of further infection from rats. Unless fleas are excluded, it is hard to determine latest exposure.

¶ This is excluding the possibility of a bite from an infected mosquito. The extended incubation period often spoken of, may be due to the fact that an infected mosquito will only be dangerous after a lapse of 12 days since the ingestion of the toxic blood. Thus a mosquito, biting a person on the 3rd day of his disease, may bite another man in, say, 14 days' time. With a superadded incubation of 13 days, the symptoms will appear in the second person 30 days (3 + 14 + 13) after they occurred in the first patient.

APPENDIX VIII.

D. T. M. REGULATIONS.

REGULATIONS OF EXAMINING BOARDS FOR DIPLOMAS IN TROPICAL MEDICINE. (D. T. M.)

DURING the past ten or twelve years, the problems of tropical medicine have occupied some of the acutest minds and most highly-trained laboratory investigators, with the result that progress has been immense and knowledge has been acquired of a sort which can be handed on to others. In addition to this, the importance of the subject has been grasped alike by the medical profession, by commercial firms engaged in tropical enterprise, and by local and home Governments. Consequently, a demand for opportunities of obtaining instruction in tropical medicine arose, and was met by the institution of two schools of tropical medicine, the one at London and the other in Liverpool. Particulars of these schools will be found below, and it is to be noted that not only have they met a demand for instruction from medical men with no knowledge of tropical disease, but they have created amongst those whose practical experience of medical work in hot countries is great a strong desire to be in a position to investigate the conditions with which they have to deal upon strictly scientific lines. The general outcome is that the whole character of medical work in the tropics and the Colonies has vastly improved. The movement owes no small measure of its success to Mr. Joseph Chamberlain, who during his tenure of office as Colonial Secretary did all in his power to encourage it. His marked interest was inherited by his successor, Mr. Alfred Lyttelton, and the change of Government has happily not altered the leanings of the Colonial Office in this direction. The net result has been that for two or three years past it has been and still is expected of every candidate for the Colonial Medical Service that he shall pass through either one school or the other before his appointment is finally approved. A further and interesting development is the institution by some of the examining bodies of a diploma in tropical medicine. The nature of work required will be gathered from the particulars given later on with regard to the diplomas granted by Cambridge and Edinburgh Universities. It seems quite possible that the possession of some such diploma may eventually be demanded by all employers who have medical appointments in the tropics in their gift. Still stronger testimony to the position which the study of tropical diseases has attained, and to the practical and scientific value to be attached to it, has within the last year been afforded by the University of London, which has now made tropical medicine one of the subjects in which a candidate can proceed to the M.D. degree (*Brit. Med. Journ.*, Sept. 1, 1906).

It is to be regretted that the London Royal Colleges did not see their way to granting diplomas in tropical medicine. The most

important reasons they brought forward (1906), against the institution of such a diploma were:—

(1) The subject is too small to merit the distinction.

(2) Other special departments might claim a similar privilege.

It will not take the student of tropical medicine many days to find the fallacy of the first objection. They have, however, granted assessors who will endorse the certificates granted by the London School of Tropical Medicine, in the case of those students who hold the M.R.C.S., L.R.C.P. diplomas.

University of Cambridge.

The Cambridge Examination takes place twice a year, in January and in August. The following are the requirements:—

Any person whose name is on the *Medical Register* is admissible as a candidate to this examination, provided—

I. That a period of not less than twelve months have elapsed between his attainment of a registrable qualification and his admission to the examination.

II. That he produce evidence, satisfactory to the Syndicate, that he has diligently studied pathology (including parasitology and bacteriology) in relation to tropical diseases, clinical medicine and surgery at a hospital for tropical diseases, and hygiene and methods of sanitation applicable to tropical climates.

As evidence of study and attainments a candidate may present to the Syndicate (1) any dissertation, memoir, or other record of work carried out by himself on a subject connected with tropical medicine or hygiene; (2) any certificate or diploma in public health or sanitary science he may have obtained from a recognised Examining Body. Such evidence will be considered by the Syndicate in determining whether he is qualified for admission to the examination, and by the Examiners in determining whether, if admitted, he shall be included in the list of successful candidates.

The examination will have reference to the nature, incidence, prevention, and treatment of the epidemic and other diseases prevalent in tropical countries. It will comprise the following subjects:—

1. The methods of pathological and bacteriological investigation. The examination of the blood. The characters, diagnosis, and life-history of animal and vegetable parasites. The examination, chemical and microscopic, of poisonous or contaminated foods and waters.

2. The origin, pathology, propagation, distribution, prevention, symptoms, diagnosis, and treatment of the epidemic, endemic, and other diseases of tropical climates, including—malaria; blackwater fever; trypanosomiasis; relapsing fever; dengue; yellow fever; plague; tetanus; beri-beri; dysentery and hepatic abscess, cholera, enteric fever, Malta fever, and specific diarrhœal affections of the tropics; diseases due to cestode and other worms; filariasis; bil

harzial disease; specific boils, sores, and other cutaneous affections; mycetoma; ophthalmic affections of the tropics; affections caused by poisonous plants and animals, and by poisoned weapons; sunstroke.

3. The general effects on health in the tropics of seasons and climate, soil, water, and food; personal hygiene, acclimatisation; principles of general hygiene, with special reference to food supplies and water supplies, sites, dwellings, drainage, and the disposal of refuse; the sanitation of native quarters, camps, plantations, factories, hospitals, asylums, gaols, pilgrim and coolie ships; principles and methods of disinfection.

The examination is partly in writing, partly oral, and partly practical and clinical. The clinical part will be conducted at a hospital for tropical diseases, at which cases will be submitted for diagnosis and comment.

Every candidate is required to pay a fee of six guineas before admission or re-admission to the examination. A candidate who, after being approved for admission, fails to present himself at the examination, will not have the fee returned, but will be entitled to present himself without further fee on one subsequent occasion.

Every candidate who passes the examination to the satisfaction of the examiners will receive from the University a diploma testifying to his knowledge and skill in tropical medicine and hygiene.

London University.

Tropical medicine is now one of the parts through which graduates of the University of London can proceed to the M.D., being number 6 of the six optional subjects of special study. Candidates for this degree in this (Branch 6) must have taken the M.B., B.S. degrees not less than two years previously, unless subsequently to that event they have either conducted a piece of original work approved for the purpose by the University, or have had such special experience as may be similarly approved; in either of these two cases the interval may be diminished to one year. All candidates alike must either have attended for not less than one academic year a complete theoretical and practical course on tropical medicine at a school approved for that purpose, or have been engaged for not less than two years subsequently to taking their degrees in hospital, official, or private practice in regions where tropical diseases prevail. The examination eventually to be passed includes a paper on General Medicine, one on Tropical Medicine, one on Tropical Pathology and Hygiene, the writing of an essay, and clinical and laboratory tests.

The Schools of Tropical Medicine approved by the University are the London Tropical School of Medicine and the Liverpool Tropical School.

University of Edinburgh.

Diploma in Tropical Medicine and Hygiene (*D.T.M.&H.*).—Graduates in medicine and surgery of this University are eligible for examination for the diploma on the expiry of a period of six months after they have obtained these degrees. They must produce evidence of having attended approved courses of instruction in practical bacteriology, including the pathogenic micro-organisms of tropical diseases, in diseases of tropical climates, including the zoological characters and the life-history of disease-carrying insects, in tropical hygiene, and in the clinical study of cases of tropical disease, and certificates of proficiency in the methods of conducting *post-mortem* examinations, and of preparing reports on them. Regulations for the diploma will be found on pp. 458-459 of the *Calendar* for 1906-7. The examinations are held in January and July. The fees for the first and any subsequent appearance are: Practical bacteriology, £1, 1s.; diseases of tropical climates, £1, 1s.; tropical hygiene, £1, 1s.; tropical clinical medicine, £1, 1s. Total, £4, 4s.

London School of Tropical Medicine.

The school buildings, laboratories, museum, library, &c., are within the grounds of the Branch Hospital, Royal Victoria and Albert Dock; Station, Connaught Road, G.E.R.

There are three courses in the year, each lasting three months, beginning October 1st, Jan. 15th, and May 1st respectively.

The laboratory, &c., are open daily, and clinical instruction is given daily in the wards of the hospitals. Certificates are granted after examination to those who complete a full course. Resident chambers are available for students, who must be qualified, or in their fifth year of medical studies.

An agreement has been concluded between the School and the Royal Veterinary College, Camden Town, by which students of either institution may attend the other; and an interchange of demonstrations has been arranged.

A Craggs Prize of £50 is awarded yearly. All information can be obtained from the Secretary, Seaman's Hospital, Greenwich.

The University of Liverpool.

Diploma in Tropical Medicine (*D.T.M.*).—Ordinance for Diploma in Tropical Medicine.

1. The University shall grant a Diploma in Tropical Medicine (written D.T.M.), which shall be awarded in pursuance of a resolution of the Senate.

2. The Diploma shall be awarded only to candidates who possess a qualification to practise medicine recognised for this purpose by the University, and who present satisfactory certificates of having

attended approved courses of study, and pass the prescribed examination.

3. Regulations shall determine :—

(*a*) The periods and courses of study.
(*b*) The subjects of examination.
(*c*) The fees to be paid for examination.

Regulations for Diploma in Tropical Medicine.—1. The examination for the diploma shall be held three times a year, at the end of the Autumn, Lent, and Summer Terms.

2. Candidates before presenting themselves for the examination, and subsequent to having obtained the recognised qualification to practise medicine, shall have attended :—

(*a*) A three months' course of study in tropical pathology and hygiene in the university.
(*b*) A course of instruction in a hospital recognised by the university, in which beds are specially reserved for tropical diseases.

3. The subjects of examination shall be :—

(*a*) Tropical pathology and parasitology.
(*b*) Tropical and applied bacteriology.
(*c*) Tropical hygiene and sanitation.
(*d*) Tropical medicine, including etiology, symptoms, diagnosis, and treatment of tropical diseases.

4. The fee for the examination for the Diploma shall be £5, 5s.

(For outline of courses, see prospectus of the Liverpool School of Tropical Medicine.)

Practical Work for the Diploma.

Both laboratory and clinical instruction can be obtained at the London School of Tropical Medicine (Connaught Road Station, G.E.R.), or at the Liverpool School of Tropical Medicine in connection with the University of Liverpool.

Messrs. Charles Baker, of 244 High Holborn, have, at my suggestion, listed two sets of microscopical specimens covering most of the ground required for the microscopical part of the D.T.M. work. These should be a most useful auxiliary to home reading, but the importance of personal "blood" work cannot be too much insisted on.

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BELL AND BAIN, LIMITED, 43 MITCHELL STREET, GLASGOW.

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